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American Journal of Diseases of Children

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OBSTETRIC PARALYSIS

ITS ETIOLOGY, PATHOLOGY, CLINICAL ASPECTS AND TREATMENT, WITH A REPORT OF FOUR HUNDRED AND SEVENTY CASES *

JAMES WARREN SEVER, M.D.

Junior Assistant Surgeon, Boston Children's Hospital; Surgeon to the House of the Good Samaritan; Consulting Orthopedic Surgeon, Cambridge Hospital

BOSTON

Obstetric paralysis, a paralysis produced during birth, is due to an injury to the nerves of the brachial plexus. The resultant paralysis is characteristic; the arm hangs vertically, the elbow extended, the forearm pronated and the whole arm inwardly rotated. The paralysis is usually flaccid.

I shall endeavor in this paper, by a review of all the literature, to give the reader the various theories as to the causes of the paralysis, as well as to offer the conclusions I have reached by a study of 470 cases. Certain experimental work which I have done, with the idea of determining clearly the etiology and pathology, will be described and conclusions drawn therefrom. The pathologic and clinical aspects of the condition will be discussed and analyzed, and the treatment, operative and nonoperative, will be dealt with under appropriate headings. Definite conclusions will be drawn from the study of about 500 cases, which will show conclusively that traction on the brachial plexus, and a resultant injury of the plexus, is the one cause of the condition.

Up to within a year or so most of us were reasonably content to accept the theory that the paralysis in these cases was due to a stretching or tearing of some of the roots of the brachial plexus, due to a forcible separation of the head and shoulders during labor. Other theories have been discussed and have been given some credence, but recently a new one has appeared. It seems that it is about time for us to take an account of stock and see which of these various ideas which have been advanced are reasonable and based on pathologic findings and clinical facts.

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* Submitted for publication June 5, 1916.

* From the Neurological and Orthopedic Department of the Children's Hospital, Boston.

Obstetric paralysis was first described by Smellie[1] in 1768, who believed the condition due to long pressure on the arm while the child was in the pelvis; but it was first brought prominently to the notice of the medical profession in 1872 by Duchenne, who described four cases in infants and attributed the condition to pressure of forceps or fingers in the axilla on the nerve trunks.

Duchenne[1] recognized that the lesion might occur in obstetric operations, such as disengaging the upraised arm in a breech or footling presentation, in delivering after version, or in making traction on the arm of the child after the birth of the head, and quotes cases to support this theory. These procedures result in direct traction on the cords of the plexus, and when force is used probably cause injuries to the nerves. It was not until 1874 that Erb[1] described the same type of paralysis in adults, since which time it has been commonly known as the Erb-Duchenne paralysis. Erb showed that pressure above the shoulder on the junction of the fifth and sixth cervical nerve roots, the so-called Erb's point, caused the characteristic grouping of the paralyzed muscles. He laid the occurrence of the paralysis especially "to the energetic application of the so-called Prague grip (Fig. 1), in which the fingers are applied like a fork over the back of the child's neck, with an after-coming head, and so endangering the integrity of the brachial plexus by energetic traction and compression.

Stransky,[1] in a most careful review of the whole literature up to 1902, presents the subject in detail and most conclusively. He reviews Smellie (1768), Danyau (1851), Guéniot (1867), and Depauls' work, the latter cited by Seeligmuller. He reports ninety-four cases from various authors whose works he has reviewed. Stransky believed that pressure as well as hard pulling in some cases was an adequate cause, especially if ether had been used and the child was asphyxiated. The following authors are quoted from Stransky's article:

Seeligmuller thought that pressure from forceps often caused hemorrhage about the plexus. Thorburn (1886) reported a case of lower arm paralysis, and believed the tearing of the nerves to be due to hyperextension of the shoulder as the arm was drawn above the head, but also ascribed it to pressure of the clavicle on Erb's point from the bad position of the arm.

Roulland (1884) gave all the various positions in which the condition could occur, and apparently believed it due to direct or indirect pressure on the plexus. Arens (1889) believed it due to hemorrhage or tearing of the nerves.

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1. Stransky: Centralbl. f. d. Grenzgeb. d. Med. u. Chir., July, 1902, p. 497, with complete bibliography to date, 1902.

Küstner (1888) advanced a theory that has been rejected at once by all other writers who have had any extensive experience with the cases, namely, that the trouble is usually due to a fracture of bones or separation of the humeral epiphysis.

Danchez (1891) believed the condition to be spontaneous, from pressure on the circumflex nerve of the arm while the child was caught in the pelvis, or that it might be traumatic from finger or instrumental pressure. He also believed that when the lower arm was

Fig. 1.—The delivery of the after-coming head, with the occiput posterior, by means of the Prague grip (Kerr).

involved the condition was one of "pseudoparalysis," as also did D'Astros (1892), that is, not a paralysis from nerve injury, but an arm held motionless on account of bruising and consequent pain, or as the result of bone injury. Gowers believed the paralysis to be due to pressure from forceps, and Weil (1896) that it was due to trauma, especially with an after-coming head. Peter thought it due to pressure of the forceps or strong lateral bending of the head, with a delayed

shoulder, or turning of the head in breech cases. Guillemot (1896) likewise supported the theory that the condition was due to compression of forceps or a strong pull; and Jolly (1896) believed it due to pressure, chiefly with an after-coming head.

Stransky quotes the experimental work of Fieux (1896), Schoemaker (1899), Stolper (1901), Küstner (1888), and Landold, as follows:

Fieux opposed Erb's views, in that Erb's point was too small and that the pressure would have to be too sharply localized, so that on the whole the theory that finger pressure or forceps could produce it was unlikely. Pressure of finger he also rejects, for there was nothing for the finger to compress the plexus against. He comes finally to traction on the upper roots as the longest side of the triangle formed by the cords of the plexus, with lateral inclination of the head, as tending to increase the distance between the head and shoulder. He produced the paralysis in rabbits by pulling the head forcibly to one side. He showed that the amount of separation which occurred between the ends of the cut roots of the brachial plexus, when the shoulder was held down and the head carried to the opposite side with as much force as is used in ordinary labors, is as follows: The two upper cords, or fifth and sixth cervical, separated from 26 to 28 mm., the third, or seventh cervical, only 12 mm., and the lower two, the eighth cervical and first dorsal, only 8 mm. The point at which the rupture occurs is from a quarter to half an inch from the point of emergence from the spinal canal near the junction of the fifth and sixth cervical roots. Fibers of the suprascapular nerve always ruptured among the first.

Schoemaker also conducted experiments on cadavers with the plexus exposed, and could always tear the fifth and sixth cervical, but never the seventh and eighth. He also thought that the clavicle could cause pressure on the plexus by having it caught between the clavicle and first rib and spine. He was opposed to the theory that pressure from the fingers caused the injury. Küstner (second paper) and Landold also did experimental work and believed the injury due to traction. Stolper agreed in the main with Fieux and Schoemaker, but rejected the possibility of pressure on the plexus in breech cases, and believed that clavicular pressure might cause the paralysis. However, he believed that stretching was the main factor.

Taking up now other authors, some of whom published their articles previous to 1902, and are quoted by Stransky, I will give comprehensive abstracts from their original articles.

Lovett[2] (1892) reports nine cases and discussed the conditions of

2. Lovett, R. W.: The Surgical Aspect of the Paralysis of New-Born Children, Boston Med. and Surg. Jour., July 7, 1892.

the labor, most of which were long, hard and instrumental. He believes that the paralysis is due to some direct injury to the brachial plexus and is generally associated with strong traction made on the head. Out of nine cases, four had the right arm affected, four the left and one case was not noted.

Carter[3] (1893) believed strongly that overstretching of the cords of the brachial plexus is the cause of the paralysis. He reported sixteen cases of his own, with an analysis of the conditions at labor, besides comparing his cases and observations with those reported by Lovett[2] and Burr. He believed that in a left occipito-anterior position the right arm would be the one affected, and in right occipito-anterior position the left arm would be the one to be injured. Nine of his own cases showed paralysis on the right and seven on the left. Burr's cases (quoted by Lovett) showed the right arm involved nine times in nine cases, regardless of position. Carter also discusses the factors of pressure of forceps on the plexus, the hook, and the finger pressing directly on the plexus in the neck, pressure of the finger in the axilla and overextension of the arm. He does not believe that these factors are essential in the production of the paralysis.

Walton[4] (1896) states that "neither the seat of the lesion nor the method of its production has been absolutely determined, but that the preponderance of evidence appears to establish the brachial plexus rather than the spinal cord as the point of injury." He discusses the relations of the position of the fetus to the paralysis of the right or left arm, and reaches about the same conclusion as Carter, except that he believes that the injury to the plexus is brought about by pressure on the plexus by its being caught between the upper edge of the clavicle and the first rib. He believes that a more careful study should be made of the positions and presentations of these children so as to determine the definite mechanics of the injury. He believes that the suprascapular nerve is independently stretched in the separation of the head from the shoulder, the distal point of fixation being either the suprascapular notch or the outer edge of the scapular spine, around which the nerve immediately passes, or both.

Haynes[5] (1897) reports three cases. He quotes Starr, who says:

> It is the pressure of the obstetrician's fingers which causes the injury in the majority of cases, and I have noticed that in 75 per cent. of the cases seen the paralysis was in the left arm, which finds its explanation in the greater

3. Carter, C. F.: Obstetrical Paralysis, with Reference Especially to the Pathology and Etiology, Boston Med. and Surg. Jour., 1893, cxxxviii, No. 18.

4. Walton, G. L.: The Etiology of Obstetrical Paralysis, Boston Med. and Surg. Jour., Dec. 24, 1896.

5. Haynes, W. H.: Obstetrical Paralysis of Infants, Brooklyn Med. Jour., May, 1897.

length of the middle finger of the hand which is doing the damage. In the act of traction there is a tendency of the obstetrician to flex the fingers, and then the tip of the finger is pressed deeply into the side of the child's neck.

This is very interesting, but hardly scientific. Haynes presents no new ideas on the subject.

Robinson[6] (1899) reports seventeen cases, in only one of which was birth reported as normal. All the others had a definite history of the labor being tedious and difficult. In eleven the presentation was cranial; in three special mention was made of difficulty in delivering the arms; four others had forceps applied. He states that J. E. Simpson has shown that the heads of boys are larger than the heads of girls, and therefore the heads of the latter would not dilate the way for the shoulders as well as the former. In his own series thirteen babies out of seventeen were girls, which would bear out this theory that there was an insufficiently dilated canal for the shoulders and that they therefore caught, or were with difficulty delivered, and in so doing there was a strain put on the cords of the plexus.

J. J. Thomas[7] (1905), Warrington and Jones[8] (1896) and Stone[9] (1900) believed the paralysis to be due to overstretching of the nerves of the plexus at birth, and Thomas reports two cases of bilateral paralysis of the lower arm type, following difficult labors with face presentation, in which he believed the injury to be the result of excessive lordosis or hyperextension in the face position, a view also concurred in by Jolly.

Bullard[10] (1907) likewise believes that overstretching of the nerve trunks is the cause of the paralysis, and that traction on the head in the axis of the body is less injurious than when the traction is made obliquely so that the head is inclined to one side when the traction is made. Rotation of the head to the opposite side also stretches the nerves. This is a factor which Walton also considers of the greatest importance. Firm resistance should be offered in order to have the force effective on the nerves, which may be supplied by a shoulder caught behind the pubes or by an after-coming head in a breech delivery (Fig. 2). Asphyxia is also a favorable condition, in that, with the child in that condition, all muscles are fully relaxed and

6. Robinson, H. B.: Traumatic Birth Paralysis of the Upper Extremity, St. Thomas Hospital Reports, 1899, xxvi.

7. Thomas, J. J.: Two Cases of Bilateral Birth Paralysis of the Lower Arm Type, with bibliography, Boston Med. and Surg. Jour., Oct. 19, 1905, cliii, No. 16.

8. Warrington and Jones: Observations on Paralyses of the Brachial Plexus. Lancet, London, Dec. 15, 1906.

9. Stone, J. S.: Injuries about the Shoulder Joint at Birth, Boston Med. and Surg. Jour., March 8, 1900, cxlii, No. 11.

10. Bullard, W. N.: Obstetric Paralysis, Am. Jour. Med. Sc., July, 1907.

their resistance is absent. Under this condition the nerves, without their usual support and protection, are more easily torn. He found that the cases generally occurred when the labor was long and difficult, when instruments were used in abnormal presentations, especially breech, and when the child was asphyxiated. In regard to the position of the occiput, it has been stated that in a left occipito-anterior position the right arm would be the one paralyzed, as the right shoulder would be the one caught behind the pubes; but this was not borne out by the few observations he was able to make, for in seventeen

Fig. 2.—Stretching of nerves by oblique traction when the shoulder is caught under the pubes.

cases of left occipito-anterior position eight of the infants were paralyzed on the right and nine on the left.

Taylor[11] (1907) states that the cause of brachial birth palsy is due to tension or overstretching of the nerves of the brachial plexus. This he has confirmed by numerous dissections and experiments on infantile cadavers. The overstretching was caused by forcible separa-

11. Taylor, A. S.: Results from the Surgical Treatment of Brachial Birth Palsy, Jour. Am. Med. Assn., 1907, xlviii, 96.

tion of the head and shoulders in vertex presentation by pulling on the head, and in breech presentation by pulling on the shoulder, by the so-called Prague method (Fig. 1). He reports the delivery of a breech case when he felt the roots of the plexus tear under his fingers, which was later confirmed by necropsy.

Osterhaus[12] (1908) likewise believes the injury to be due to overstretching.

Bailey[13] (1908) believes the condition concerns the obstetrician more than it does any other practitioner. He states that while pressure is the generally accepted cause, he is of the opinion that overstretching and traction on the plexus is the real one. He states that

Fig. 3.—Stretching of nerves by oblique traction in delivering the posterior shoulder when caught on the perineum (Bumm).

if the axis of the head is drawn away from the long axis of the body by 30 degrees, the cords of the plexus are drawn to the danger point. This is liable to happen in vertex presentations to hasten the delivery of the shoulder, and in breech presentations to hasten the delivery of the after-coming head. It may also happen in extraction by forceps,

12. Osterhaus, Karl: Obstetrical Paralysis, New York Med. Jour., Nov. 7, 1908.

13. Bailey, P.: Brachial Birth Palsy, Bull. Lying-In Hosp., New York, March, 1908.

and in spontaneous birth the delivered head by its own weight may cause traction on the plexus (Figs. 3 and 4).

Frazier and Skillern[14] (1911) speak of the older theory that the brachial plexus injuries were caused by the plexus being crushed or squeezed between the clavicle and first rib, or transverse processes of the cervical vertebrae. Subsequent observations, however, have proved that in all cases the essential element in the causation is traction on the nerves.

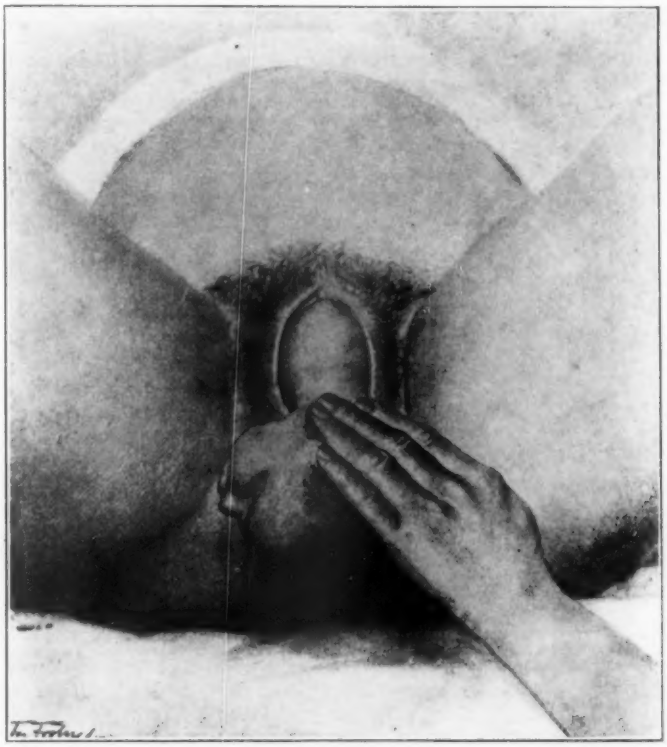

Fig. 4.—Separation of head and shoulder, with shoulder caught behind the pubes (Nagel).

Lange[15] (1912) believes the paralysis is due to a tearing of the capsule of the shoulder joint, which at first limits motion because of pain and then from habit. He was the first to suggest the theory which T. T. Thomas has taken up, that the condition is purely secondary to a shoulder joint injury. It should rightly be called Lange's theory.

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14. Frazier and Skillern: Suprascapular Subcutaneous Lesions of the Brachial Plexus Not Associated with Skeletal Injuries, Jour. Am. Med. Assn., 1911, lvii, 1957.

15. Lange: München. med. Wchnschr., No. 26, 1912.

Frauenthal[16] (1912) believes also in the overstretching theory and reports cases, but is rather optimistic as to his results.

T. T. Thomas[17] (1914), in an interesting theoretical discussion of the problem, based on a study of nine cases averaging 6.5 years, concludes that the paralysis is secondary to a primary traumatic dislocation of the shoulder occurring at birth, associated with a tear in the joint capsule and a consequent involvement of the plexus in the exudate, practically Lange's theory, as given above. He does not explain why the exudate always avoids the major portion of the plexus in the region of the shoulder joint, or why it practically always works its way at least two inches above the clavicle and picks out the junction of

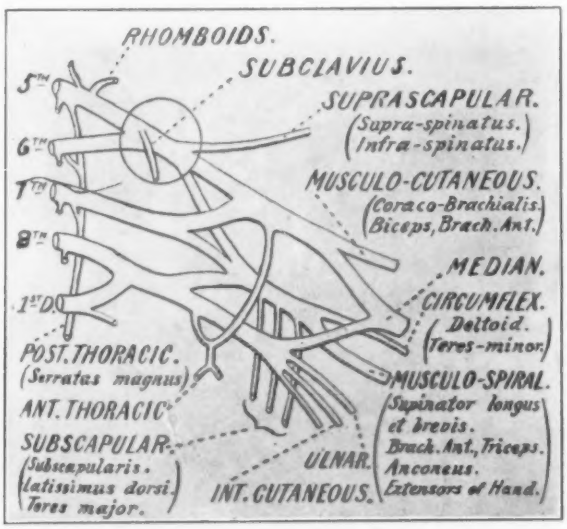

Fig. 5.—Brachial plexus, showing Erb's point. The subscapular nerve in this illustration comes off below Erb's point, but generally arises from the fifth nerve above its junction with the sixth cervical root.

the fifth and sixth cervical nerves to produce the characteristic paralysis. This theory of his, which is purely philosophical, is ingenious, but not reasonable, nor is it based on clinical or pathologic evidence. Erb's point is small and it requires definite injury at this point to produce the characteristic paralysis, as well as injury above this point on the fifth cervical root to produce the paralysis of the supraspinatus and infraspinatus from trauma to the suprascapular nerve which comes off the fifth cervical just above or below Erb's point (Fig. 5).

16. Frauenthal: Erb's Palsy, Am. Jour. Obst., 1912, lxv, No. 4.

17. Thomas, T. T.: The Relation of Posterior Subluxations of the Shoulder Joint to Obstetrical Palsy of the Upper Extremity, Ann. Surg., 1914.

Fairbank[18] (1913) is another believer in the traction theory, and he reports forty cases, thirty-two of which were vertex presentations and seven breech, which rather refutes Tubby and Sherren, whom he quotes and who believe that it occurs equally in the two presentations. He also states that long, difficult labors, in which forceps were used, predisposed to the injury (Figs. 6, 7 and 8).

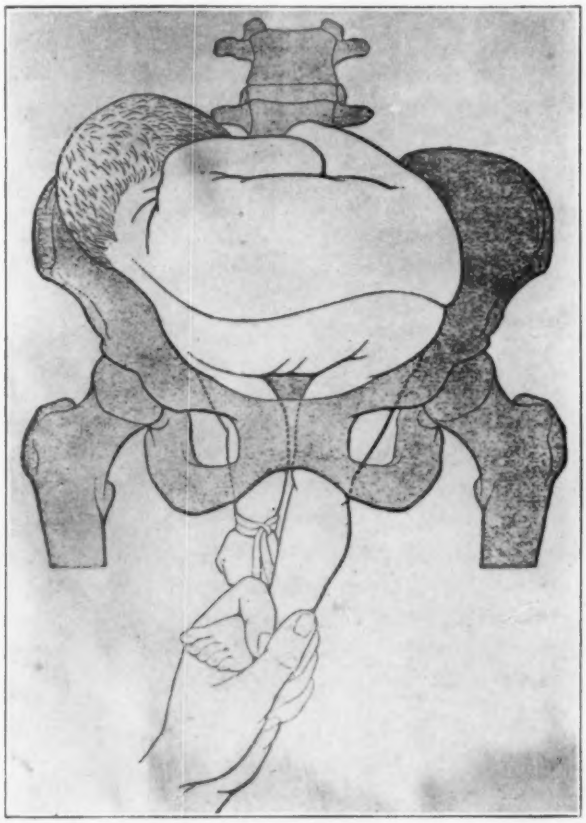

Fig. 6.—Version in dorso-anterior position, first stage; a difficult labor, predisposing to injury (Faraboeuf and Varnier).

Peltesohn[19] (1914) has found a number of cases which he reports as "false birth palsies." He describes typical end-results of cases of obstetric paralysis. He states that the condition is due to injury of the upper epiphysis of the humerus at birth. In true Erb's paralysis there is no disturbance of the epiphysis.

18. Fairbank, H. A. T.: Birth Palsy; Subluxation of the Shoulder Joint in Infants and Young Children, Lancet, London, May 3, 1913.

19. Peltesohn, S.: Injuries of the Upper End of the Humerus in Birth Palsies, Berl. klin. Wchnschr., June 22, 1914, p. 1162.

Gaugele[20] (1914) states that so-called obstetric paralysis is not a true paralysis, and the cause of the condition is an injury to the capsule and soft parts with subsequent contraction. Injury to the epiphysis or other injury is not uncommon. He evidently bases his conclusion on the study of four cases, and is not familiar with the work of other observers.

Van Neck[21] (1914) believes that other conditions than injury to the plexus may simulate obstetric paralysis, such as epiphyseal injuries of the head of the humerus, congenital developmental errors of the plexus, and shoulder turning resulting in a tear of the capsule. These all present definite clinical pictures, and by Roentgen ray and careful clinical examinations the diagnosis should be made easy and not confused with obstetric paralysis.

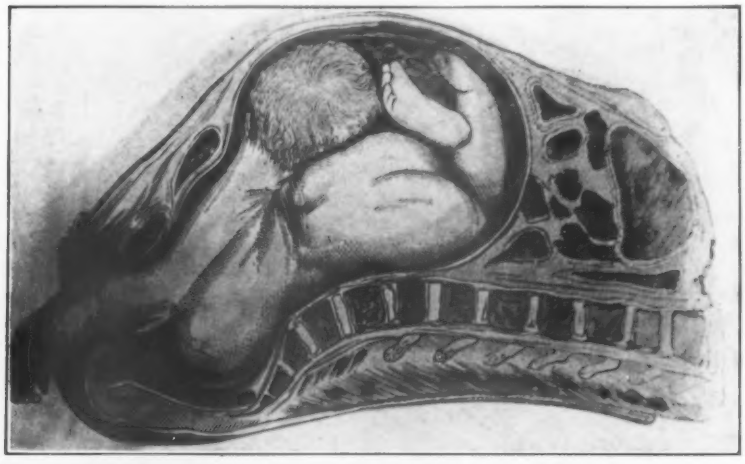

Fig. 7.—Neglected shoulder presentation; section through frozen corpse; a difficult labor, predisposing to injury (Chiara).

Gordon[22] (1914) believes also in the traction theory, as well as the theories of direct pressure on the plexus by the obstetrician's finger, the hook, and pressure by the clavicle and transverse processes.

Platt[23] (1915) is also a follower of the traction theory, and bases his ideas on five cases. He quotes other authors, including Lange and Vulpius, the former believing in the laceration of the capsule theory

20. Gaugele, K.: So-Called Obstetrical Paralysis of the Arm, Ztschr. f. orthop. Chir., 1914, xxxvi, Nos. 3 and 4.

21. Van Neck: Congenital or Obstetrical Lesions of the Shoulder and Brachial Plexus, Jour. méd. de Bruxelles, 1914, No. 11.

22. Gordon, A.: An Unusual Form of Birth Palsy, Jour. Am. Med. Assn., 1914, lxiii, 2282.

23. Platt, H.: Birth Palsy, Brit. Med. Jour., May 8, 1915.

and the latter in the theory of epiphyseal displacement as causes of the paralysis and subsequent deformity.

Darling[24] (1915), in an extensive study of the various lesions of the brachial plexus and a discussion of the various theories, accepts the ones based on definite nerve findings and pathology, and believes that traction on the cords of the brachial plexus is the generally accepted one in view of clinical and experimental evidence.

Fig. 8.—Forcible separation of head and shoulder in a shoulder presentation, putting the plexus on a stretch, which is almost sure to result in injury to the nerves (Kleinwächter).

Sharpe[25] (1916) has demonstrated that direct injury of the nerves always occurs, as shown by operation on the plexus in fifty-six cases;

24. Darling, H. C. R.: Med. Jour. Australia, Oct. 9, 1915.
25. Sharpe, W.: The Operative Treatment of Brachial Plexus Paralysis, Jour. Am. Med. Assn., 1916, lxvi, 876.

in every case finding definite pathologic evidence of injury. He believes the injury is caused by overstretching of the plexus at birth, due generally to a prolonged, forcible separation of the head and shoulder by lateral extension during a difficult labor.

This rather hasty review of practically all the literature on this subject from the etiologic point of view shows that the majority of observers incline toward the traction theory, which is in turn definitely supported by pathologic and clinical evidence. It is not questioned that fracture and epiphyseal displacement occur, and that they may be associated with an injury to the brachial plexus as a separate entity, but that they are the one cause of the usual type of birth palsy cannot be accepted. I have seen a number of cases of fracture of the upper end of the humerus occurring at birth which simulated brachial palsy, but on careful study were properly diagnosed. The after-course was quite different from that seen in brachial plexus injuries.

PATHOLOGY

There are generally two well-recognized types of paralysis seen. The more common one consists of a lesion which involves the fifth and sixth cervical roots and the suprascapular nerve and produces a paralysis of only the muscles of the upper arm, with the exception of the supinators. This type is known as the upper arm type. The less usual type, the so-called lower arm, a whole arm type, is the result of injury not only to the fifth and sixth cervical roots, but the seventh and eighth and possibly the first thoracic as well. Here the whole arm is flaccid; there is a wrist-drop and paralysis of the small muscles of the hand. There rarely occurs the pure lower arm type of paralysis without any involvement of the upper cords of the plexus, the so-called Klumpke's paralysis, several cases having been reported by J. J. Thomas, Jolly, Guillemot, Seeligmuller, Thorburn, Raymond, Comby and Danchez. These cases show a paralysis usually the result of stretching of the plexus from overextension of the head in cases of face presentation, and due to injury to the lower cords of the plexus, namely, the seventh and eighth cervical roots. They may at times be bilateral. It is in this type that one often sees inequality of the pupils, owing to the fact that the sympathetic fibers from the deep cervical ganglionic plexus enter the spinal cord through the first dorsal and at times through the eighth cervical roots. Injury therefore to these roots leads to an unopposed action of the motor occuli nerve.

Pathologically, in the milder cases the stretching or tearing forces result in a greater or less degree of hemorrhage or edema into the nerve sheaths. In others there may be a rupture of the perineural sheath, accompanied by hemorrhage into the substances of the nerve trunk, associated with a tearing apart or separation of the nerve fibers.

TABLE 1.—AUTHORS WHO HAVE REPORTED CASES, THE DATES OF THEIR REPORTS AND NUMBER OF CASES REPORTED

Authors	Date	Number Cases Reported	Authors	Date	Number Cases Reported
Duchenne	1872	4	Schoemaker	1899	2
Nadaud	1872	3	Haslinger	1899	2
Erb	1874	2	Bollenhagen	1899	1
Ducouneau	1876	2	Robinson	1899	17
Sceligmuller	1877-1882	8	Steiner	1900	1
Roulland	1884	1	Thomas, H. M.	1900	3
Thorburn	1886	1	Maygrier	1901	1
Arens	1889	1	Stolper	1901	1
Henoch	1890	1	Peter, cited by Stransky.	1902	2
Danchez	1891	4	Oppenheim, cited by Stransky	1902	1
Cited by Danchez:					
Budin	....	1	Koster, cited by Stransky	1902	4
Babinski	....	1			
Monnier	....	2	Schultze, cited by Stransky	1902	1
Burr	1892	8	Thomas, J. J.	1905	2
Lovett	1892	9	Bullard*	1907†	178
D'Astros	1892	1	Murphy	1907	1
Bailly and Onimus, cited by D'Astros	....	1	Taylor, A. S.	1908	10
			Osterhaus	1908	2
Comby, cited by D'Astros	....	1	Rhode	1909	1
Carter	1893	16	Frauenthal	1912	4
Hochstetter	1893	1	Fairbank	1913	40
Weil	1896	1	Lange	1913	17
Fieux	1896-7	2	Thomas, T. T.	1913	9
Jolly	1896-7	3	Peltesohn	1914	5
Guillemot	1896	12	Gaugele	1914	4
Walton	1896	2	Gordon	1914	1
Haynes	1897	3	Van Neck	1914	3
Warrington and Jones	1896	2	Platt	1915	5
Cibert	1897	2	Sharpe	1916	56
Plauchu	1898	1			
Fifty-eight authors. Total number of cases reported to date 457. Number of cases reported in this paper					470

* The Bullard cases are included in this paper, which actually gives a total of reported cases of from 279 to 470.
† Forty-three in detail.

This latter condition leads, of course, to permanently impaired function and the formation of scar tissue in the nerve track. In the more severe cases of the upper arm type there is a partial or complete division of the fifth and sixth cervical roots, which leads to a more permanent form of paralysis than usual, and the formation of a more extensive area of scar tissue.

The force producing these lesions is variable and so the lesions are variable. The nerve roots are often frayed out inside the sheath instead of being torn across evenly, and in this way the lesion may be incomplete at any given cross section of a nerve, but involves different fibers at different levels. This scar tissue contracts in time, and not only effectually prevents the regeneration of the nerves, but may by its contraction press on and destroy the few fibers which may have escaped the original injury.

When there has been a complete or partial evulsion of the fifth and sixth cervical nerves from the spinal cord the condition pathologically is as follows: The spinal meninges over the affected area are thickened, fibrous and adherent to the cord. The affected side of the spinal cord is smaller than normal, and the injured areas of the cord may be invaded by the scar tissue. The anterior horns may be more or less disturbed, with a reduction in the number of the cells, which show various stages of degeneration; adjacent nerve tracks are more or less damaged. The anterior and posterior roots, as well as the brachial plexus on the affected side, are smaller than normal. Changes also take place in the cerebrum analogous to those found after amputation, such as a reduction of the Betz cells and a gross lesion of the fibers in the motor, intermediate and precentral area (Robinson[6]).

The other type, known as the lower arm or whole arm type, is the result of either a lesion involving all the nerves of the plexus, or, in the distinctly lower arm type, in which the lower arm and hand are alone involved, the so-called Klumpke's paralysis, in which the lesion probably involves the eighth cervical and first dorsal roots alone. This type generally results from traction applied in a breech case with the arm extended, or to traction in the axilla in a vertex presentation. It may be seen also in adults, when the first dorsal root is overstretched, as evidenced by some of the cases reported by T. T. Thomas.[17] Pathologically, the conditions are similar to those seen in the other types, depending on the severity of the injury. No case in which operation has been performed has failed to show a definite pathologic lesion of the brachial plexus, definitely corresponding to the muscles involved.

Danyau (quoted by Stransky[1]) in 1851 showed by necropsy that the nerves of the plexus had been torn and were surrounded and

invaded by scar tissue. Boyer[26] also reports necropsy findings, and states that the "opposite side of the spinal cord was distorted and otherwise altered by the injury and resulting fibrotic changes." Practically all observers, especially those who have operated in these cases, have found definite changes in the plexus due to injury and scar tissue formation. Among these, for detailed study, may be mentioned Fairbank, Warrington and Jones, Osterhaus, J. J. Thomas, Stone, Taylor and Prout.[11] Prout's description of the pathology is classic and will be quoted freely as follows:

Prout states that the nerve sheath in any overstretching process must give way before the nerve itself, as it supports the nerve. When the sheath is torn, as it always is in cases of birth palsy, the arterioles belonging to it and supported by it are ruptured, and a hemorrhage into the substance of the nerve and its sheath results. These facts are of the greatest importance, since they determine the ultimate extent and final character of the lesion. Were it not for the obstructive features of the repair process in the nerve sheath, we might expect a more or less complete recovery in the vast majority of cases.

Four pathologic specimens showed on study the following conditions: The usual seat of the lesion was at the junction of the fifth and sixth cervical nerves. The perineural sheath presented many old dense pigment deposits, the site of old hemorrhages. In some portions the perineural sheath was buckled inward on the nerve fibers, strangulating them and preventing their regeneration. Evidences of strangulation were present not only at these points, but also in the nerve fibers underlying these pigment deposits. There was an obliteration of the myelin sheath above and below. In the more severe cases the strands of the plexus involved came to an abrupt termination in a mass representing an old organized hemorrhage. In these cases there was a severing of the nerve fibers, which were often thrown into folds for some distance from the primary lesion. Repair of the nerve sheath takes place before regeneration of the nerve fibers, and if this has buckled inward on the nerve bundles following relief of tension, the nerve fibers are inevitably going to be strangulated and their regeneration prevented.

AUTHOR'S EXPERIMENTS[27]

The author, by numerous dissections on infantile cadavers, has shown that traction and forcible separation of the head and shoulder puts the upper cords, the fifth and sixth cervical roots of the brachial plexus, under dangerous tension. This tension is so great that the two upper cords stand out like violin strings. Any sudden force

26. Boyer, G. F.: Proc. Roy. Med. Soc., Neurol. Sect., 1912, p. 31.

27. Work done in the Laboratory of Surgical Pathology, Medical School of Harvard University, by courtesy of Dr. E. H. Nichols, director.

applied with the head bent to the side and the shoulder held would without question injure these cords. Further observation shows that forcible abduction and elevation of the arm and shoulder put the lower cords of the plexus, the eighth cervical and first thoracic on a stretch, and when much force is applied it may well lead to a tear, rupture or other injury to these segments. This condition is seen in breech cases, with arm extended. It may also follow sudden strain when the arm

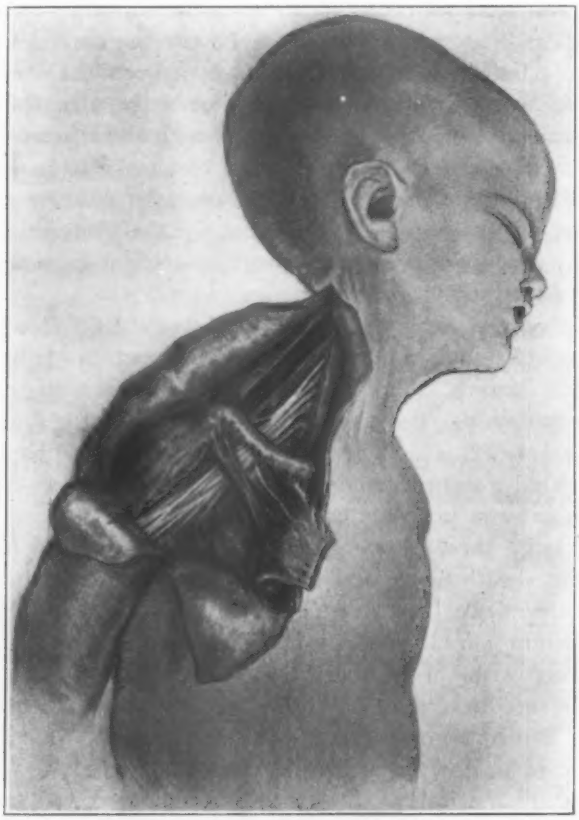

Fig. 9.—Dissection of the brachial plexus in a baby, with the head and shoulder in natural relation.

is elevated, such as the so-called hostler's paralysis, caused by the sudden elevation and strain of the arm which occurs when a hostler holds a rearing horse. With the shoulder held and the head carried to one side, with the clavicle intact, considerable force was necessary to injure the plexus. The suprascapular nerve always snapped first, apparently for the reason that it had not so much freedom of play as the others. Even with considerable force the fifth and sixth cervical

nerves could not be completely torn across at Erb's point, but frayed out inside the sheath, following a partial tearing or rupture of the sheath, which always gave way first. In some cases there could be produced an evulsion from the spinal cord of the fifth and sixth cervical roots.

With the clavicle removed, the whole weight of the shoulder came practically directly on the plexus, and less force had to be exerted to cause an injury, which under these conditions was generally greater in extent, but presented the same general characteristics. It was most

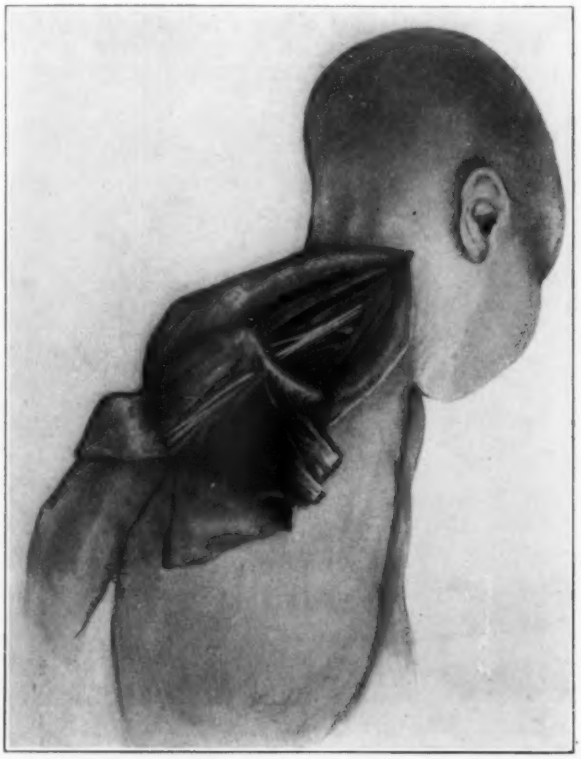

Fig. 10.—Same as Figure 9, with the head and shoulder forcibly separated.

difficult to put the eighth cervical and first thoracic roots on a stretch unless the arm was abducted or hyperextended with great force.

With the clavicle intact there was apparently always enough room, even with the arm elevated and hyperextended forcibly, between the clavicle and plexus so that direct pressure from the intact clavicle on the plexus did not seem a possible cause of the paralytic condition. A fractured clavicle of course allows the weight of the shoulder to drag on the plexus, and so predisposes to greater injury from traction.

Rotation of the head combined with forcible abduction apparently does not increase the degree of tension greatly, certainly not enough to cause additional damage. In no case, even with all the force I could apply with my hands, could I rupture the joint capsule, or even separate the humeral epiphysis. Neither could I dislocate the head of the humerus. The clavicle can be broken without great force, but fracture of the other bones which go to make up the shoulder joint is practically impossible. Most birth fractures occur in the clavicle, or in the humerus, at about the junction of its upper and middle third. Stone[9] states in the experimental work which he did that the humeral epiphysis could be easily separated, but I failed to confirm this.

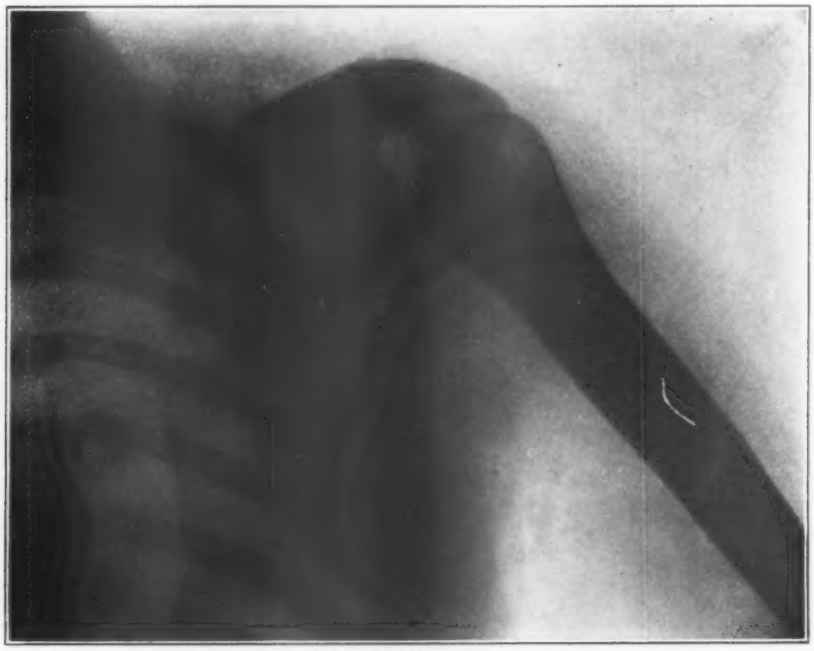

Fig. 11.—Roentgenogram of the shoulder of a patient 18 years of age. Note the hooking of the acromion and subluxation of the head of the humerus, with elevation and outward rotation of the scapula.

At birth the shaft of the humerus is nearly wholly ossified, but the two extremities are cartilaginous. The scapula at birth is largely osseous, with the exception of the glenoid fossa, the coracoid and acromial process, and the posterior border and inferior angle, which are still cartilaginous. It is on account of these conditions that fractures in these regions at birth are practically nonexistent. It is not possible to produce a paralysis of the Erb type by the fracture of any bone but the clavicle.

In order to get a clear idea as to what happened to an exudate from a ruptured capsular ligament of the shoulder, in studying Lange's theory, I injected the shoulder joints of several infants with methylene blue, and then caused a rupture of the anterior portions of the joint capsule. The infants were then allowed to lie in a preserving solution on their backs for several weeks, following which time a dissection was made. In no case did the methylene blue go above the clavicle, but completely surrounded and invaded the plexus in the axilla. This would in life lead to a paralysis of the whole arm below the joint, but would in no way affect the nerves above the clavicle, and in no case would there be the typical picture of obstetric paralysis, that is, paralysis of the fifth and sixth cervical nerves. As I have stated before, why the exudate should leave the nerves alone in immediate

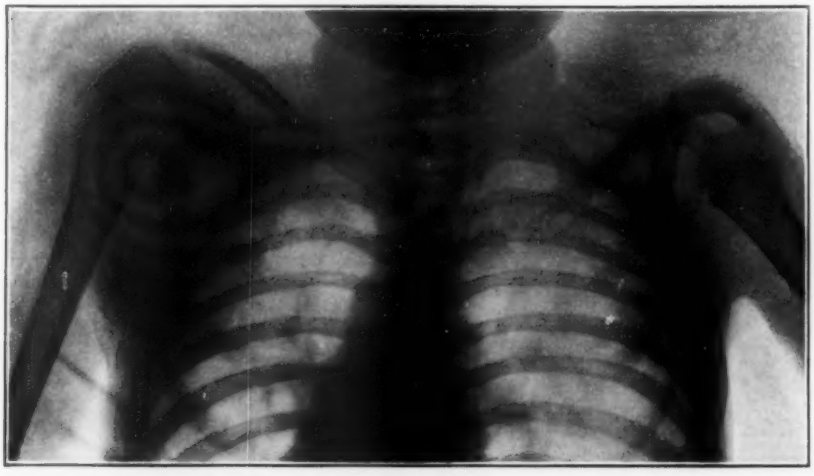

Fig. 12.—Roentgenogram of the shoulders of a boy, aged 16, showing the same characteristics as Figure 11. The normal shoulder is shown on the left.

proximity of the shoulder joint and seek out Erb's point, the junction of the fifth and sixth cervical segments, at least two or three inches above the clavicle, Lange, Thomas and others have not made quite clear. It evidently does not happen. Why also should the suprascapular nerve always be involved, which generally arises from the fifth cervical at about Erb's point? One thing impressed me, and that was the evident vulnerability of the upper cords of the plexus under any degree of traction and I was surprised that the paralysis was not of much more frequent occurrence (Figs. 9 and 10). Figure 9 shows dissection of a baby's plexus with the head and shoulders in natural relations. Figure 10 shows the head and the shoulder forcibly separated. Note the folding together of the cords of the whole plexus, especially the fifth and sixth cords.

Roentgen-Ray Findings.—One hundred and nine of the recently observed 170 cases of obstetric paralysis have had roentgenograms taken of both shoulders on one plate. These patients have varied in age from 2 days to 18 years. In only two cases had there been fracture, one of the clavicle and one of the upper third of the humerus. Both fractures had healed without incident. These cases are classified in Table 2 according to their ages at the time the roentgenogram was taken.

A study of the roentgenograms taken in these cases shows the following conditions:

In the first year there is usually nothing seen of bony deformity. There may be a slight posterior subluxation of the shoulder joint,

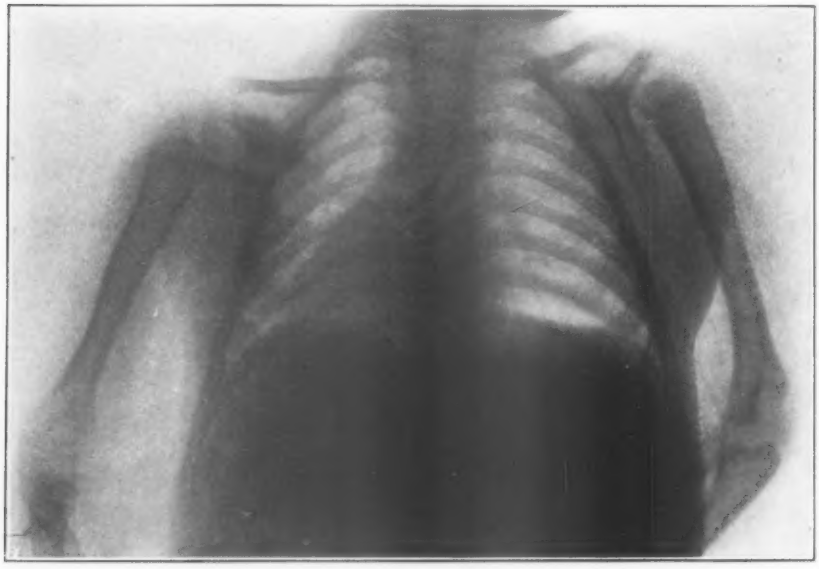

Fig. 13.—A younger patient than that shown in Figure 12. An outward displacement of the scapula is seen on the right.

but there is never any acromial deformity evident by roentgenogram or clinically. No case has been observed in which the epiphysis has been displaced so far as could be seen by comparison with the normal shoulder. The epiphysis, as well as the shaft of the humerus, is always smaller than the unaffected side, which condition is undoubtedly due to atrophy from disuse. The scapula is practically always elevated and outwardly rotated, due apparently to the pull of the intact inward rotators and the levator anguli scapulae.

As time goes on and the child gets older, one begins to see increasing evidences of bony deformity, occasionally more joint subluxation

than at first, increasing outward displacement and elevation of the scapula, and acromial deformity. The deformity of the acromion consists of a bending downward and forward or a hooking of its outer end, which apparently, having no bony resistance to meet as normally in the head of the humerus, projects downward in front of the subluxated and inwardly rotated head. This hooking seems to vary directly with the degree of posterior subluxation and inward rotation of the humerus and tends to increase as the child gets older, provided

TABLE 2.—CONDITION SHOWN BY ROENTGENOGRAM IN 109 CASES

Ages, Years	Subluxation of Joint	Acromial Deformity	Elevation and Displacement Outward of Scapula	Joint Apparently Normal
*	11		16	15
1	6	3	7	5
2	5	1	5	5
3	4	3	5	4
4	4	4	6	4
5	6	6	6	
6	3	4	5	2
7	5	3	6	
8	2	1	3	
9	4	4	4	
10	3	3	3	
11	2	2	2	1
12	1	1	1	
13	2	1	2	
14	3	2	3	
15	1	1	1	
16	1	1	1	
17				
18	1	1	1	

* Age from 1 day to 2 years.

subluxation is present. No case has been observed in which there has been a total subluxation or dislocation of the shoulder joint backward. The clavicle usually is shorter and its curves are more acute than its normal fellow (Figs. 11, 12 and 13).

Clinical Findings.—When the child is first seen, if within a few days or weeks after birth, the following picture is classic. The arm lies limp at the side, extended and inwardly rotated, with complete inability to abduct, elevate, outwardly rotate or supinate. The muscles

paralyzed in the typical upper arm type are as follows: Deltoid, supraspinatus, infraspinatus, teres minor, biceps, supinator longus, and occasionally the serratus magnus, coracobrachialis and supinator brevis. The arm cannot be actively flexed at the elbow, but as a rule the lower arm is not affected so far as flexion and extension of the wrist and flexion and extension of the fingers go (Figs. 14 and 15).

The greater part of the motor nerve supply to these paralyzed muscles depends on one root alone, although fibers from more than one root can be traced to individual muscles of the arm. The root distribution of the nerves of the brachial plexus is as follows (Quain[28]):

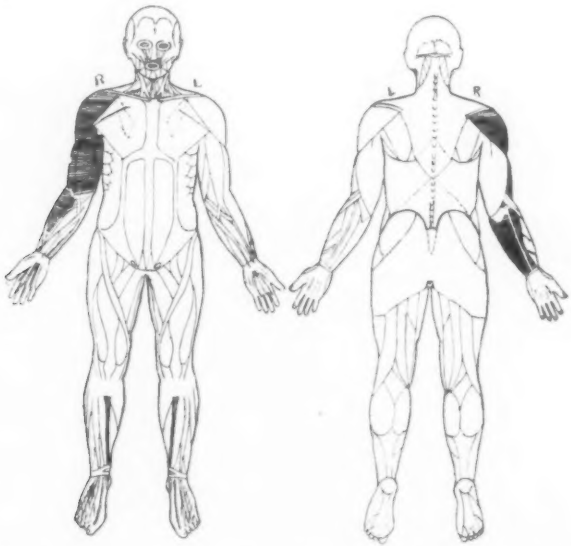

Fig. 14.—Typical upper arm type of obstetric paralysis in a girl of 10 weeks; the muscles shown in white are normal, those in dark shading are paralyzed.

The fifth cervical supplies the levator scapulae, rhomboidei, serratus magnus, supraspinatus, infraspinatus, teres minor, subscapularis, deltoideus, biceps brachii, brachialis anticus (?), pectoralis major (?), teres major.

The sixth cervical supplies the serratus magnus (?), supraspinatus (?), infraspinatus (?), teres minor, subscapularis, teres major, deltoideus, pectoralis major, biceps brachii, brachialis anticus, pronator teres, flexor carpi radialis, supinator longus and brevis, extensor carpi radialis, abductor opponens and flexor brevis pollicis.

The seventh cervical supplies the serratus magnus, pectoralis major and minor, latissimus dorsi (?) teres major, coracobrachialis, triceps

28. Quain: Anatomy, iii, Part 2, p. 354.

brachii anconeus, flexor sublimis digitorum (?), flexor profundis digitorum (?) flexor longus pollicis (?) pronator quadratus, extensor radialis, extensores digitorum, extensor carpi ulnaris (?) abductor opponens (?) and flexor carpi pollicis (?).

From the eighth cervical are supplied the pectoralis major and minor, latissimus dorsi, triceps, anconeus, flexores digitorum, flexor carpi ulnaris, pronator quadratus, adductor pollicis, interossei, abductor flexi brevis and opponens, and abductor minimi digiti.

From the first dorsal are supplied the pectoralis major and minor, flexores digitorum, flexor carpi ulnaris, pronator quadratus.

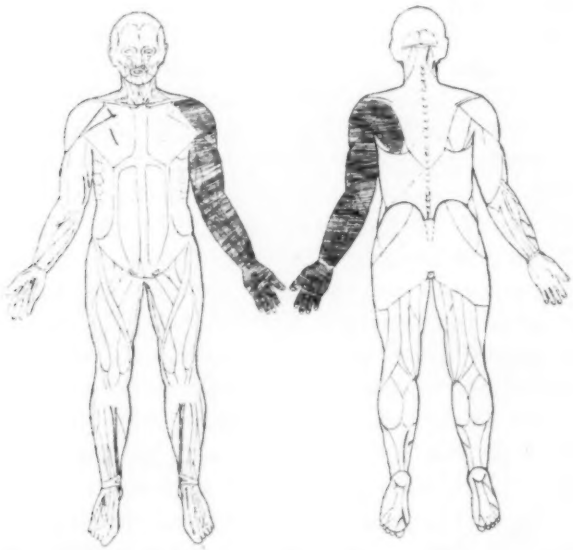

Fig. 15.—Typical lower or whole arm type of obstetric paralysis in a girl of 3 months; the muscles shown in white are normal, those in dark shading are paralyzed.

Tracing back the nerves to their origin, we find the following facts:

From the outer cord: The external anterothoracic follows back to the sixth, seventh and fifth (?) cervical; the nerve to the coracobrachialis to the seventh cervical; the musculocutaneous to the fifth and sixtr cervical; the outer head of the medianus to the sixth and seventh cervical.

From the posterior cord: The upper subscapular is traceable to the fifth and sixth cervical; the lower subscapular to the fifth (?) and sixth cervical; the circumflexus to the fifth and sixth cervical, and the musculospiralis to the sixth, seventh and eighth cervical (Fig. 5).

It should be noted that a number of these muscles have more than

one source of supply. Expressed in terms of motion the condition is as follows:

Flexion of the elbow is carried out by the fifth cervical; extension of the elbow by the seventh cervical; pronation of the hand by the sixth cervical; supination of the hand by the fifth cervical; flexion of the wrist by the eighth cervical, and extension of the wrist by the seventh cervical.

In the upper arm type then, the nerves involved are the suprascapular, from the fifth cervical root and outer cord of plexus, going to the supraspinatus and infraspinatus muscles. The musculocutaneous from the fifth and sixth cervical roots and outer cord of the plexus, going to the coracobrachialis, biceps and brachialis anticus. The circumflex from the fifth and sixth, and possibly the seventh and eighth and posterior cord of the plexus, going to the deltoideus and teres minor. The musculospiralis from the fifth, sixth and seventh, and also possibly some fibers from the eighth cervical and posterior cord of the plexus, going to the supinator longus and brevis, brachialis anticus, triceps, anconeus and extensors of hand.

The fact that in the upper arm type practically the only muscles supplied by the musculospiralis which are paralyzed below the elbow are the supinators goes to show that either the injury is not extensive or that the nerve root supply is well divided. No two diagrams of the brachial plexus among all that I studied were alike. The cut of the one shown is the most satisfactory, and, as far as I could tell, the most usual type of formation of the plexus (Fig. 5).

In order to get this definite and constant paralytic muscle grouping, the injury would have to be located at about the junction of the fifth and sixth cervical nerve roots, just above the point of origin of the suprascapular nerve. This junction point is called Erb's point, from his classic description of the type of paralysis seen following injury at that point.

The inability to raise or abduct the arm at the shoulder is due to the paralysis of the deltoideus and supraspinatus. Outward rotation cannot be accomplished because of the paralysis of the infraspinatus and teres minor, and the arm cannot be internally rotated owing to the internal rotators, namely, the teres major, subscapularis and latissimus dorsi, being already fully contracted, due to lack of opposition.

The arm cannot be flexed at the elbow, owing to the paralysis or weakness of the biceps, brachialis anticus, coracobrachialis and supinator longus; and supination cannot be carried out owing partially to the inward rotation in which the arm is held and the weakness or paralysis of the biceps and supinator longus and brevis.

In regard to sensation, it may be stated that it has been impossible

TABLE 3.—Analysis of 394 Cases of Obstetric Paralysis, Showing Condition at Different Ages, Age When Signs of Recovery Were Noted, and in the Older Patients the Condition on Entrance to Clinic

Age	Complete Paralysis		Partial Recovery		Total Recovery		Elevates to Shoulder or Above		Wrist Drop	Paralysis of Hand, Partial or Complete
	Upper	Lower	Upper	Lower	Upper	Lower	Upper	Lower		
2 weeks	11	..	..	..	..	..	..	..	..	..
3 weeks	5	..	4	..	..	..	1	..	..	..
5 weeks	7	..	7	..	..	..	..	..	..	..
6 weeks	3	1	2	1	..	..	..	..	1	1
7 weeks	7	..	7	2	..	..	1	..	..	..
2 months	8	2	14	..	1	..	6	..	..	..
10 weeks	1	..	7	..	..	..	4	..	..	..
3 months	3	3	27	1	..	..	10	1	2	..
4 months	..	..	12	1	..	..	6	1	1	1
5 months	..	1	9	..	..	..	7	..	..	..
6 months	..	..	15	1	..	..	15	1	1	..
7 months	..	..	10	..	..	..	9	..	..	..
8 months	..	3	11	3	..	..	9	1	3	3
9 months	..	..	12	..	..	..	11	..	..	..
10 months	..	..	3	..	..	..	2	..	..	..
1 year	..	..	48	7	1	..	47	5	6	4
2 years	..	..	27	5	..	..	26	3	5	3
3 years	..	1	16	2	..	..	15	1	3	3
4 years	..	..	19	2	2	..	19	2	2	1
5 years	..	..	7	2	..	..	7	2	3	1
6 years	..	..	12	2	1	..	12	1	2	2
7 years	..	1	6	2	..	..	6	2	3	2
8 years	..	..	8	..	1	..	7	..	..	..
9 years	..	..	4	..	..	..	4	..	..	..
10 years	..	..	3	..	1	..	2	..	..	..
11 years	..	..	6	1	..	..	7	..	1	..
12 years	..	..	2	..	..	..	2	..	..	..

in the early cases to determine any changes from the normal, on account of the age of the patient. Likewise, electrical reactions have not been carried out, for this examination would mean anesthesia, which did not seem justifiable in such young children, when one already had all necessary data.

During the first week, in the early cases, the child may cry if the arm is handled or moved, especially in abduction, but this soon disappears. In one or two cases there has been some swelling and tenderness noted by palpation over the plexus above the clavicle. This condition, however, apparently had no connection with the degree of paralysis present. The hand grasp is usually good and the child flexes and extends the wrist and fingers well. The later developments in the upper arm cases, as the child grows and gets older, with or without exercises and massage, are as follows: The persistence of the inward rotation and adduction deformity, the so-called policeman's tip position; the inability in most cases to fully or freely supinate; the inability to get the hand to the mouth without raising the elbow, due to inability outwardly to rotate; the inability to put the hand to the head or behind the back.

In the lower arm type all these conditions hold, besides the additional ones due to the paralytic conditions of the lower arm and hand, resulting generally in a useless dangle arm.

Atrophy of the muscles in these cases of obstetric paralysis is never very marked, except in some cases of the lower arm type. One never sees the extreme atrophy so noticeable in cases of infantile paralysis. This lack of marked atrophy is undoubtedly due to the fact that the nerve impulses are rarely fully blocked and that the muscles practically never, except in rare cases, wholly lose their entire enervation. Some normal nerve impulses pass through the scar tissue at the site of the lesions, owing to incomplete destruction or injury of the nerve, and so keep the muscle tone up to a certain point. There is always a definite shortening of the arm, however, in all cases, due probably as much to nerve injury as lack of use.

Referring to Table 3, which shows the detail of the cases reported, we may note that there are 400 of the upper arm type of paralysis. These in the main showed the conditions mentioned above.

SUBSEQUENT DEVELOPMENTS

Whole Arm Type, Lower Arm Type.—There were seen sixty-four cases of this type in this present series. In this classification those cases which showed any nerve involvement beyond that usually shown by an injury of the fifth and sixth cervical roots were placed. These cases represented those injuries mainly to the whole of the plexus, or at least the seventh and eighth cervical and the first dorsal roots.

Pupillary inequality and narrowing of the palpebral fissure were not unusual with this type. Wrist-drop was the usual condition, associated with the usual inability to supinate and the additional inability to extend the lower arm. Paralysis of the flexors and extensors of the wrist and fingers were common, associated with paralysis and atrophy of the intrinsic muscles of the hand. Often the proximal phalanges are hyperextended, and the distal ones flexed, due to the paralysis of the interossei or lumbricalis manus muscles. There is, of course, no power to grip and the fingers cannot be moved. There is usually ulnar displacement or adduction of the hand. These cases, almost without exception, represent severe tearing injuries to the roots of the

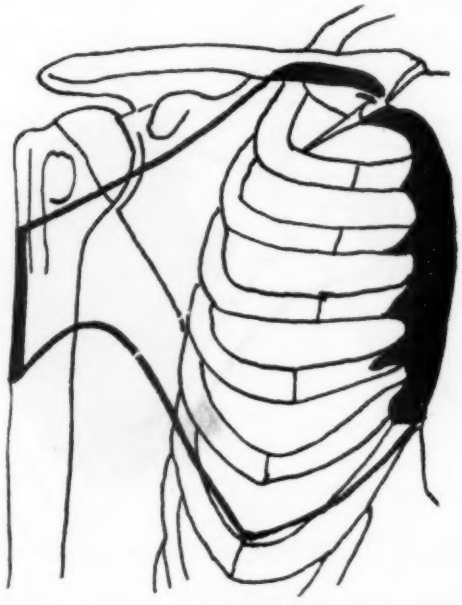

Fig. 16.—Pectoralis major of right side; outline and attachment areas (Gerrish).

plexus, and although some of the muscles may recover in part, particularly the upper arm and shoulder groups, the lower arm ones practically never recover, even after attempted operative repair of the plexus. It is in these cases that sensation is more apt to be impaired than in the usual upper arm type. A not uncommon type seen is one showing simply a wrist-drop, associated with the usual picture of upper arm paralysis and evidence of injury to the fifth, sixth and seventh cervical roots. These cases, as far as results go, should be classed with the simple upper arm type. Few cases have been recorded in which the two lower roots alone have been involved. These have been reported fully by J. J. Thomas.[7]

The complications may be divided into two classes, early and late. The early complications are those accompanying the paralysis and present at birth. The following may be mentioned:

Facial paralysis is usually mild and on the same side as the paralyzed arm and is probably from forceps pressure on the facial nerve.

Fracture of clavicle is not rare.

Separation of epiphysis of the head of the humerus may occur, but no case is noted in this series; it might be grouped under the pseudoparalysis of D'Astros and Danchez.

Dislocation of the humerus sometimes is present, usually infraspinatus. This complication is not noted in this series, but is recorded by other observers.

Fracture of the upper third of the humerus may also occur.

Fig. 17.—Subscapularis of right side; outline and attachment areas (Gerrish).

As late complications the following may be mentioned:

Posterior subluxation of the humerus is common and due to contraction of unparalyzed pectoralis major, subscapularis and teres major (Figs. 16 and 17).

Hooking of the acromion may occur, as has been already noted above.

Anterior subluxation of the humerus, due to the pull of the contracted pectoralis major and the stretching of the subscapularis, is not uncommon.

Contraction of the biceps and the brachialis anticus, leading to some degree of permanent flexion deformity at the elbow and occasionally dislocation of the head of the radius, may occur.

An analysis of Table 4 may be of interest. In the first place, there is no reason to expect any difference in regard to the sex, unless one

is ready to accept Simpson's theory that girls' heads, being smaller, and so not dilating the canal sufficiently, would subject them to a more difficult labor, and so to a greater percentage of occurrence of injury to the brachial plexus. These figures, representing by far the largest number of cases so far reported, and outnumbering all others reported by all observers, do not confirm his theory.

The right arm was affected 272 times and the left 186, about 68 per cent. in favor of the right arm. This bears out Sharpe's figures in his series of fifty-six operative cases. Nine babies had both arms affected.

The types of paralysis differed, the most usual one being the so-called upper arm type, 400 being recorded, as against the so-called lower or whole arm type, in which, besides the fifth and sixth cervical cords being injured, the seventh and eighth cervical and first dorsal

TABLE 4.—Conditions Existing at the Time of Birth

Boys	235	
Girls	236	
Total		471
Right arm affected	272	
Left arm affected	186	
Both arms affected, upper arm type	2	
Both arms affected, lower or whole arm type	1	
Both arms affected, type not noted	6	
Upper arm type	400	
Lower or whole arm type	64	
Difficult labor	418	
Ether used	363	
Forceps used	317	
Normal labor	32	
Asphyxiation of child	102	
Head presentation (including face)	219	
Breech presentation (including foot and version)	66	
Position not known	186	
Fractured clavicle	14	
Arm broken	3	
Cord around neck and arm	2	
Cord around neck	2	
Pupils unequal	16	

were injured. Of the latter type sixty-four cases were recorded. In nine cases with both arms affected the lower or whole arm type of paralysis showed generally.

It has been conceded by practically all authors that a difficult labor was a predisposing factor in the causation of paralysis. In this series 418 cases were definitely recorded as long, laborious and difficult; 363 at least had ether and 317 had forceps used; thirty-two were apparently normal labors and 102 were recorded in which the child was asphyxiated.

All the conditions noted above imply the application of force combined with great muscular relaxation of the child, conditions peculiarly favorable for the production of such an injury. A moderately large number, it is recorded, had the head delivered naturally, but the shoulders stuck, and at that time force was applied.

In regard to the presentations, 219 at least were vertex or face presentations and sixty-six were breech. The latter classification includes versions and footlings. In 186 the position was not recorded, but a large majority of these were probably vertex. These figures do not bear out either Tubby or Sherren (quoted under Fairbank[18]), who state that the paralysis occurs equally in head or breech presentations. Fairbank's own figures refute this also, for he reported in forty cases thirty-two vertex and seven breech. These figures cover 285 cases of the author's in which the presentation was definitely known.

The other conditions occurring at birth may be noted in Table 4, and I want to add a word about only one of them, namely, that of unequal pupils. This condition is probably overlooked in some cases, and is a most important symptom, in that it means that through injury to the cervical sympathetic there may be definite injury to the plexus either of the lower cords, the eighth cervical or first dorsal, which have communicating bands with the cervical sympathetic, or injury in the spinal cord itself to the fibers of the sympathetic system. The prognosis in these cases is usually not so good as in those which do not show this sign.

TREATMENT

As to treatment, these cases at once resolve themselves into two divisions, namely, those to be treated with massage and exercises, principally those of the upper arm type; and those to be treated by operation on the plexus, usually those of the lower arm type. Unless the early treatment has been adequate, the upper arm type will also come to operation, not for plexus repair, but to correct contraction deformities. This operation, which I have devised, will be spoken of later.

At first, in order to prevent contraction of unparalyzed muscles, it seems best to put the arm at rest in such a position that the muscles cannot become contracted. This may be done by holding the arm in a plaster cast, or by the use of a light wire splint, in an abducted, elevated and outwardly rotated position, with the hand supinated. This position can be maintained between massage and gymnastic treatments, and insures a better subsequent position of the arm. It also takes the drag off the paralyzed muscles, allowing them to regain their strength more quickly, and prevents subsequent shoulder joint deformity, such as subluxation and acromial hooking and overgrowth.

Massage and exercises are of the greatest importance and should be done daily if possible. It is most unwise to allow a child to become obsessed with the fact that it has an arm which cannot be used. Exercises which have been described in detail by J. J. Thomas[29] are

29. Thomas, J. J.: Obstetrical Paralysis with Especial Reference to Treatment, Boston Med. and Surg. Jour., April 2, 1914, clxx, No. 14.

most satisfactory, and have been developed during the past twenty years in the neurologic department of the Children's Hospital. The treatment should be continued for several years at least, and if contractures develop in the subscapularis and pectoralis major, they must be divided before any further range of action in the arm is to be hoped for.

In regard to the operation on the plexus in the usual upper arm type of case, it might be said that in the experience of this clinic it has not been found necessary. In the lower arm type of cases the situation is quite different, but it cannot be too strongly emphasized that no operation on the plexus will be of any great use in restoring functional activity to the arm, unless contracted and restricting muscles are divided, and careful after-treatment persisted in for a long period.

In regard to the operative treatment on the plexus in the lower arm type of case, it may be stated that it has been done a number of times without any benefit. The plexus in all cases was found to be so badly torn and so bound down and invaded by scar tissue that any kind of repair was impossible. In spite of the work done by A. S. Taylor,[11] Stone,[9] Fairbank[18] and others, there has been no case as yet which has shown an anatomic or physiologic cure, or even a marked improvement. This may be due to the fact that in the first place the plexus was impossible to repair, and secondly, granted that the plexus repair was in part possible, the muscular contractions and joint deformities were not recognized and properly treated, without which the attempt to obtain plexus repair would be a waste of time and effort.

The following operation was devised, following suggestions made by Fairbank.[18] It differs from Fairbank's operation in that the shoulder joint is not opened. Opening this joint leads to adhesions of the capsule, which are troublesome and fatal to the best functional results. In addition, I have found that complete division of the pectoralis major is always advisable, in that it is practically always tightly contracted, and so holds the arm adducted and prevents abduction and outward rotation. The subscapularis tendon can usually be easily found with the arm abducted and outwardly rotated after the division of the pectoralis major, and can be divided without opening the joint capsule.

OPERATION

An incision is made situated on the anterior aspect of the arm and extending from the clavico-acromial joint to a point below the lower edge of the pectoralis major tendon. The incision is carried down between the deltoid and clavicular portions of the pectoralis major, tying or retracting the cephalic vein. The tendon of the pectoralis major is isolated and divided on a director. Turning the cut pectoralis

Figure 18

Figure 19

Fig. 18.—The skin incision and the incision between the deltoid and pectoralis major.

Fig. 19.—The pectoralis major cut and the deltoid and the pectoralis major retracted; the long head of the biceps is in the floor of the wound.

Figure 20

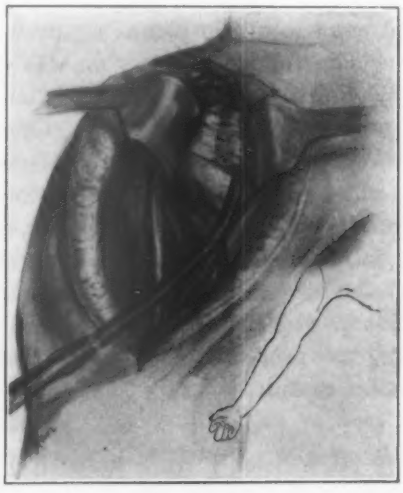

Figure 21

Fig. 20.—The joint capsule with the insertion of the subscapular tendon on inner aspect.

Fig. 21.—A sound is passed under the tendon of the scapularis, the arm being abducted and rotated out to its limit.

major back and retracting the deltoideus gives a good view of the long head of the biceps and the joint capsule, as well as the short head of the biceps and coracobrachialis. The arm is now abducted and outwardly rotated, bringing into view the transverse fibers of the tendon of the subscapularis at its point of insertion into the joint capsule at its inner and anterior aspect. This tendon is isolated and a sound or other blunt instrument is passed under it, and it is then divided. In this way not only is the pectoral divided, which, when contracted, prevents abduction, but also the subscapularis is divided,

Fig. 22.—E. L., October, 1915. Before operation on right arm.

which, when contracted, prevents outward rotation. It is better to divide the subscapularis by this method, rather than to open the joint capsule, after Fairbank's method, for it does not lead to subsequent adhesion of the capsule to the joint cartilage and consequent loss of motion.

After these two structures have been cut, outward rotation and abduction will usually be found to be perfectly free. In case either is at all restricted, the coracobrachialis or the short head of the biceps may be found to be tight, and the partial division of these structures

will always lead to full freedom in outward rotation and abduction. If the head of the humerus is blocked by the hooking downward of the acromion in front of it, so that the posterior subluxation cannot be fully reduced, an osteotomy can be easily done on the acromion, through the upper end of the original incision.

If there is an anterior subluxation of the joint, which occurs rarely, the pectorialis major is the only muscle which needs to be divided. A division of the subscapularis would only tend to increase a deformity already present. The pectoralis major and deltoideus are then joined with interrupted catgut sutures, and the skin closed by a continuous catgut suture. The arm is then put into a plaster cast extending from the crest of the ilium to the tips of the fingers, the arm being abducted, elevated, outwardly rotated and the hand supinated. This cast should be worn only about two weeks, at the end of which time baking,

Fig. 23.—E. L., April 12, 1916. Three months after operation on right arm.

massage and exercises should be started and continued daily for several months. After two or three weeks a wire splint may be substituted for the cast, in that it is lighter and more comfortable (Figs. 18, 19, 20 and 21).

RESULTS OF OPERATION

Twelve patients so far have been operated on. The first few operations were done by Fairbank's method and the patients were kept in plaster for the length of time advised by him, namely, three months. It is too long. Although they were improved, it has required persistent effort and considerable difficulty to restore motion in the shoulder joint, besides muscle strength, and the results were not commensurate with the time and effort expended.

Recently, since I have been doing the operation described above, combined with early treatment, that is, by giving massage, manipulation

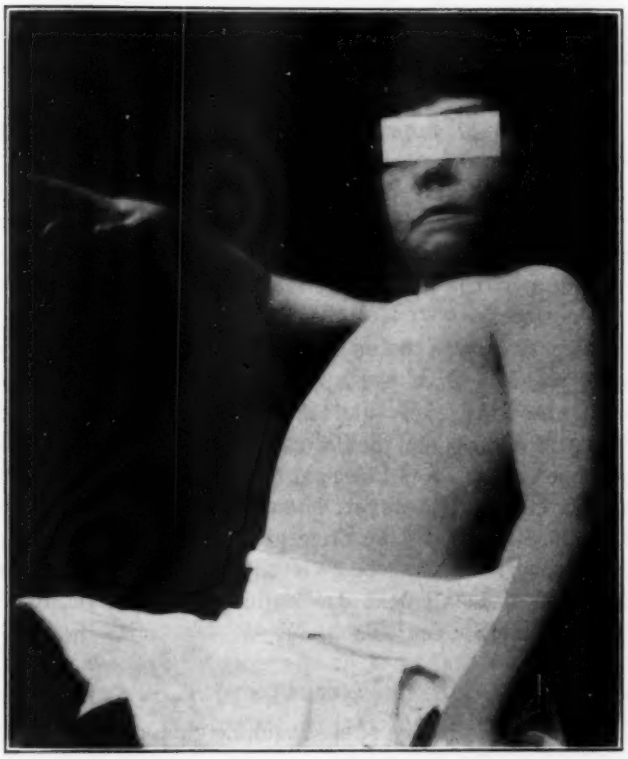

Fig. 24.—R. B., Nov. 17, 1915. Right arm before operation.

Fig. 25.—R. B., April 26, 1916. Five months after operation on the right arm.

and exercises, at the end of two weeks the results have been much better. This is what might have been expected. Once the contractions are divided, long fixation is obviously unnecessary (Figs. 22, 23, 24 and 25).

PROGNOSIS

The prognosis in all upper arm type of cases is good, provided the case is watched from the start, and treatment properly carried out. The patients are practically all able to raise the arm to the shoulder level and can use the hand and lower arm well, except for varying degrees of supination. Abduction and outward rotation are rarely regained without division of the contracted muscles, provided they have been allowed to contract.

In the lower arm type the outlook is not so good, although many of the patients regain use of the upper arm in spite of the persistent paralysis of the lower arm and hand. These cases should all be explored for repair of the plexus as far as possible, but even then very little hope can or should be held out to the parents. The general principle of treatment, however, should be carried out over a long period of time. Much can be done along orthopedic lines for these patients, and they should not be generally neglected as they have been in the past, with the statement that nothing can be done, or that they will get well of themselves (Table 3).

CONCLUSION

Obstetric paralysis is due to a stretching or tearing of the cervical roots of the plexus brachialis. It occurs in boys as frequently as in girls. It occurs more often on the right than on the left side.

The upper arm type is much more frequent than the lower arm type. It affects both arms very infrequently.

It is practically always associated with a difficult labor, in which ether and forceps have been used and force has been applied. Not uncommonly is the baby asphyxiated.

Head presentations show the larger percentage of occurrences of both types of cases.

It may rarely be associated with fracture of the clavicle, but is not the result of a fractured humerus or a dislocated shoulder joint.

The prognosis for a useful arm is good in the upper arm type and bad in the lower arm type.

A STUDY OF THE TOPOGRAPHY OF THE PULMONARY FISSURES AND LOBES IN INFANTS

WITH SPECIAL REFERENCE TO THORACENTESIS *

J. CLAXTON GITTINGS, M.D., GEORGE FETTEROLF, M.D.
AND
A. GRAEME MITCHELL, M.D.
PHILADELPHIA

In the course of a general study of the anatomy of the infant's chest it seemed to us desirable to attempt to determine accurately the relation of the fissures of the lung to the bony framework of the thorax. We felt that carefully obtained data would be valuable not only for comparison with similar relations as they exist in the adult, but also as a stimulus toward greater accuracy in the clinical diagnosis of pleuropulmonary disease.

A careful search of the standard textbooks and of the periodical literature which has appeared since 1905 shows nothing in reference to the course of the fissures in infants. Symington[1] states that the only gross difference between the position of the lungs in children and in adults lies in the fact that in the former the anterior margins of the lungs are not as closely approximated as in the adult, in other words, that the right and left lungs at times do not meet or overlap under the manubrium sterni. He makes no mention of any difference in the course or relations of the fissures. On the other hand, there are numerous references pertaining to adults. Many of these doubtless are mere copied repetitions, but the conflicting descriptions point to the conclusion that the course of the interlobar fissures is fixed only within comparatively wide limits.

The right lung, it will be remembered, normally possesses two fissures. One of these, the oblique, starts above and behind and runs downward, outward and then forward; the second, the horizontal fissure, begins at a variable point above the middle of the oblique fissure and passes almost horizontally in toward the sternum.

The left lung normally possesses but a single fissure, the oblique, which corresponds to the like structure on the right side.

Various anomalous fissures exist quite frequently, the most common, according to Shaffner,[2] being found on the lower surface of the

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* From the Laboratory of Anatomy of the University of Pennsylvania.

1. Symington, J.: The Anatomy of the Child, E. & S. Livingstone, Edinburgh, 1887, p. 64.

2. Shaffner, quoted by Piersol, George A.: Human Anatomy, J. B. Lippincott Co., Philadelphia, 1907, p. 1846.

lower lobe. This is sometimes merely a shallow indentation, but at others is a deep fissure sharply delimiting the inner portion of the lower lobe.

In addition to anomalous fissures, there are also found at times variations in the normal fissures. Of the latter the horizontal is the one most frequently found to be atypical and at times it may be absent. Conversely, in the left lung, a transverse fissure occasionally is found dividing it into three lobes.

That the course of the oblique fissures in adults is far from constant may be inferred from the descriptions found in the literature.

Right Lung: It begins at the second dorsal vertebra (Morris,[3] Gray,[4] Deaver[5]), the second or third (Spalteholtz[6]), the third (Fraenkel[7]), the third or fourth (Corning[8]), the fourth (Bickham,[9] McClellan,[10] Davis,[11] Chauffard[12]), the fourth interspace or the fifth rib (Rochard[13]), the fifth rib or the fourth or fifth interspace (Piersol[14]). It terminates at the fifth intercostal space or the sixth rib (Rochard,[13] Gray,[4] Davis,[11] Morris,[3] Deaver,[5] Piersol[14]), the seventh rib (Bickham,[9] McClellan,[10] Fraenkel[7]), the eighth rib (Chauffard[12]).

Left Lung: It begins at the second dorsal vertebra (Gray,[4] Morris,[3] Deaver[5]), the second or third dorsal vertebra (Spalteholtz[6]), the third rib (Bickham,[9] Fraenkel,[7] McClellan[10]), the third or fourth rib (Corning,[8] Davis[11]), the fourth interspace (Chauffard[12]), the third to the fifth rib (Piersol[14]). It terminates at the sixth rib according to most of these authorities, except Chauffard,[12] who finds it at the seventh interspace.

Authors' Technic.—Our own observations are based on dissections

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3. Morris, Sir Henry: Human Anatomy, edited by J. P. McMurrich, P. Blakiston's Sons & Co., Philadelphia, 1907, Part 5, p. 1295.

4. Gray, Henry: Human Anatomy, edited by Robert Howden, Lea & Febiger, Philadelphia and New York, 1913, p. 1299.

5. Deaver, John B.: Surgical Anatomy, P. Blakiston's Son & Co., Philadelphia, 1903, iii, 409.

6. Spalteholtz, quoted by Dietlen, H.: Ergebn. d. inn. Med. u. Kinderh., 1913, xii, 197.

7. Fraenkel, A.: Therap. d. Gegenw., 1910, li, 337.

8. Corning, quoted by Dietlen, H.: Ergebn. d. inn. Med. u. Kinderh., 1913, xii, 197.

9. Bickham, Warren S.: Operative Surgery, W. B. Saunders Co., Philadelphia, 1908, p. 772.

10. McClellan, George: Regional Anatomy, J. B. Lippincott Co., Philadelphia, 1892, i, 267.

11. Davis, G. G.: Applied Anatomy, J. B. Lippincott Co., Philadelphia, 1913, p. 198.

12. Chauffard: Rev. gén. de clin. et de thérap., 1914, xxviii, 420.

13. Rochard, quoted by Gary, M. P.: Arch. de méd. et pharm. mil., 1910, lvi, 104.

14. Piersol, George A.: Human Anatomy, J. B. Lippincott Co., Philadelphia, 1907, p. 1859.

of the formaldehyd-hardened bodies of fourteen infants, varying in age from 6 weeks to 15 months. Of these, six were under 4 months of age and six were 4 months or older; in two the exact age was unknown, but it approximated 3 to 6 months. In order to expose the fissures in exact relation to the ribs, all of the soft tissues covering the ribs, sternum and spine, as well as the intercostal muscles and the parietal layer of the pleura, were carefully removed. Enough of the muscles of the shoulder girdle were left to insure stability of the clavicles, scapula and upper ribs. To allow of access to the interior of the chest the sternum was freed, the attachments of the costal cartilages

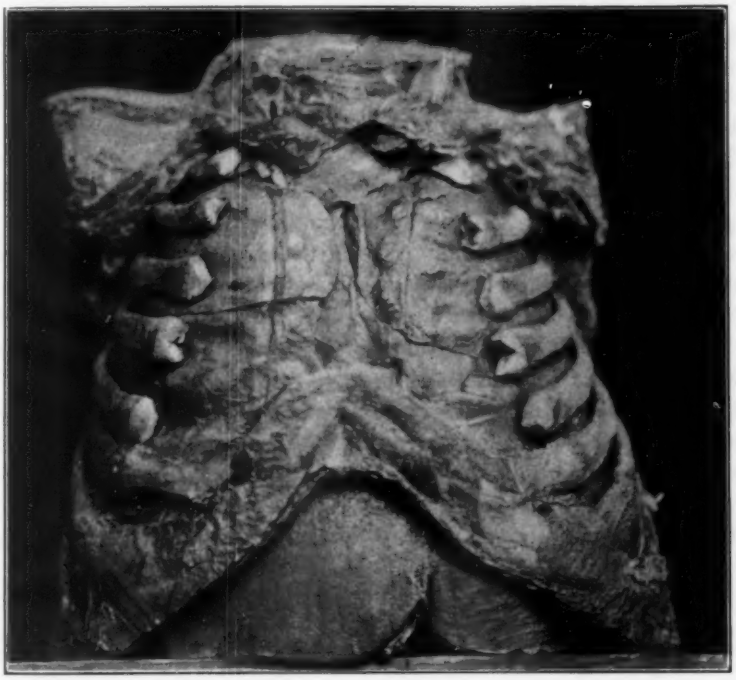

Fig. 1.—Photograph of a thorax showing the method of preparing the specimens for studying the relations of the pulmonary lobes and fissures in situ.

being noticed in order to insure accurate replacement. The spine was allowed to remain untouched as a permanent support. As the bodies had been injected with formaldehyd, the manipulations essential to study in no wise disturbed the relationship of the structure as it had existed at the time of death. The photograph (Fig. 1) shows to a certain extent the result of one of the dissections, but it is impossible to depict in this manner the entire course of the fissures in relation to the ribs. By gentle separation of the ribs, however, this could accurately be determined in every case.

Unfortunately, the removal from our specimens of the soft tissues of the chest destroyed all the ordinary clinical landmarks of the axillary region. This is to be regretted, since the point of origin of the horizontal fissure from the oblique is a matter of considerable practical interest, mainly because it is the most obvious point of attack in dealing with a collection of pus between the upper and middle lobes.

An attempt was made to compensate for this loss of landmarks by marking the ribs with an indelible pencil or by nicking them with a knife before removing the pectoral and latissimus dorsi muscles. Neither of these methods was successful, as the pencil marks became smeared in the course of the subsequent dissection and the incisions into the ribs rendered them so fragile that the attempt was abandoned. In place of this we established the midpoint of the axilla by bisecting a horizontal line drawn from midsternum to the spinous process at a point just below the angle of the scapula. This we have called the midthoracic line.

In twenty-eight children in the wards of the Children's Hospital, varying in age from 15 days to 5 years, we compared the relation between this midthoracic line and the usual landmarks of the axilla, namely, the midaxillary and postaxillary lines. The latter landmarks were first located and carefully marked, and then the measurements for the midthoracic line were taken and compared with these markings. It was found that the midthoracic line invariably lay posterior to the midaxillary line, a distance varying from 0.5 to 2.5 cm. (average of all cases, 1.27 cm.), while the postaxillary line lay posterior to the midthoracic line in every case but one, the distance varying from 0.5 to 1.5 cm. (average of all cases, 0.92 cm.). In the one exception, a child of 4 years, the midthoracic line lay 0.4 cm. posterior to the midaxillary line and exactly in the postaxillary line.

In general, we may say, therefore, that the midthoracic line lies approximately midway between the midaxillary and postaxillary lines. As the determination of the clinical landmarks by the eye is not so accurate or so reliable as the actual measurement by the tape, the latter procedure possesses distinct advantages.

Authors' Findings.—Our studies have shown us that the course of the pulmonary fissures in the infant may be described as follows:

Right Lung, Oblique Fissure: The origin is from the third to the fifth rib at the spine. In ten out of fourteen the point of origin was at the fourth rib or fourth interspace, in three at the third rib or third interspace and in one at the fifth rib, the average of fourteen cases being the fourth rib.

The course is downward and forward, crossing the midthoracic line between the third rib and the sixth interspace. Of the fourteen cases, one crossed at the third rib, two at the fourth rib, five at the

fifth rib or fifth interspace and six at the sixth rib or sixth interspace, the average of fourteen cases being the fifth rib.

The termination is from the sixth to the seventh rib just posterior to the costochondral junction. Four were at the line of the costochondral junction and ten were from 1 to 3 cm. posterior to it. Of the fourteen cases, six terminated at the sixth rib, two at the sixth interspace and six at the seventh rib, the average of fourteen cases being the sixth interspace.

Right Lung, Transverse Fissure: The origin is from the third interspace to the sixth rib in the line of the oblique fissure. Of the fourteen cases, one was at the third interspace, seven were at the fourth rib or fourth interspace, five at the fifth rib or fifth interspace and one at the sixth rib, the average of fourteen cases being the fourth interspace.

The course is almost horizontal, either beneath the fourth rib or the interspace above or below it.

The termination is at the edge of the sternum from the third rib to the fourth interspace. Of the fourteen cases, six were at the third rib or third interspace, seven were at the fourth rib or fourth interspace and one was anomalous, terminating in the second interspace 3 cm. to the right of the sternal border, the average of thirteen cases being the upper border of the fourth rib.

Left Lung, Oblique Fissure: (In one case this fissure began at the fifth rib, passed downward more vertically than usual, crossing the midthoracic line at the eighth rib. Thence it continued forward beneath the eighth rib to its costochondral junction, an anomalous type. This is excluded from the summaries of this side, leaving but thirteen.)

The origin is from the second to the fifth rib. Of the thirteen cases, four were at the second rib or second interspace, two at the third rib or third interspace, seven at the fourth rib, the average of thirteen cases being the third interspace.

The course is downward and forward, crossing the midthoracic line behind the third to the sixth rib. Of the thirteen cases, one was at the third rib, four were at the fourth rib or fourth interspace, four at the fifth rib and four at the sixth rib. Average of thirteen cases, fifth rib.

The termination is from the sixth to the seventh rib at or posterior to the costochondral junction. Seven were at the line of the costochondral junction and the remainder about 1 cm. posterior to it. Of the thirteen cases, seven were at the sixth rib and six at the seventh rib, the average of thirteen cases being the sixth interspace.

To sum up, we find that the oblique fissure of the right lung in infants begins at the fourth rib (from the third to the fifth as extremes), crosses the midthoracic line between the fourth and sixth

ribs and terminates between the sixth and seventh ribs, at or just posterior to the costochondral junction. In adults, according to the literature, the origin seems to be somewhat higher, although the tip of the dorsal vertebra, which is used as a landmark by some authors, almost corresponds to the rib below it, the second dorsal vertebra is equivalent to the third rib, etc. The point of termination seems to be the same in both infants and adults.

The oblique fissure of the left lung in infants begins at the third interspace (from the second to the fifth ribs as extremes), crosses the midthoracic line between the fourth and sixth ribs (as on the right side) and terminates between the sixth and seventh ribs, at or just posterior to the costochondral junction. In adults the origin and termination are described as being about the same as our findings in the infant.

The transverse fissure in infants begins at the line of the oblique fissure between the fourth and fifth ribs (from the third to the sixth rib as extremes) just in the midthoracic line or more often slightly posterior to it, and passes horizontally inward to terminate at the junction of the fourth rib or interspace and the sternum. In adults the exact beginning of the transverse fissure usually is not clearly described by the authorities consulted, but apparently it corresponds very closely to our findings in the infant.

It will be seen, therefore, that the differences between the fissures in adults and infants are slight and unimportant and that in both they show equally as marked variation in their point of origin, course and termination. Apparently this variability is dependent on no discoverable factors. We state this as the result of an attempt to establish some relationship between the anatomic characteristics of the chest and the location of the fissures. In this part of our study we investigated the following points in regard to the chest: (1) the general shape; (2) the anteroposterior and transverse diameters; (3) the circumference; (4) the subcostal angle; (5) the obliquity of the ribs, determined (a) at the spine and (b) in the midthoracic line. To these we added (6) the size of the liver (with special relation to the fissures of the right lung).

The result of this study was entirely fruitless and our conclusion is as stated above, namely, that these factors have no constant effect on the position of the fissures. We were somewhat surprised at this, as it would seem at first thought that the great difference between the shape of the chest in the adult and in the infant might readily influence the course and relationship of the fissures at the different ages. Apparently, however, the change in the shape of the chest goes pari passu with the development of the lung, with the result that the relation of fissure to rib or to interspace remains practically unchanged.

An important point is made by Dietlen,[15] who calls attention to the fact that descriptions of the course of the fissures refer only to the relations at the surface of the lung, since the planes formed by the inward projection of the fissures through the lung traverse various lines. This is of some importance in operations on interlobar collections, although the perfection of Roentgen-ray technic has rendered so much aid in determining localized collections of fluid as to minimize the importance of other aids to diagnosis. This is especially fortunate since the normal relations of the fissures are profoundly modified by interlobar collections.

We have been impressed with the fact that students and interns usually disregard the position of the fissures and fail to take advantage of the aid which may be gained from observing their relations. For example, an area of dulness involving the region of the right apex and sharply following the line of the horizontal fissure of the right lung usually will prove to be croupous pneumonia or its delayed resolution, rather than a tuberculous pneumonia. Again, an area of dulness which follows and lies below the line of the oblique fissure usually will be due to a pneumonia rather than to a collection of fluid in the pleural cavity, or a tuberculous pneumonia; while the gradual extension of the border of dulness beyond the limits of the lower half of the oblique fissure will point to an empyema, rather than to the extension of a pneumonic process, as the latter is more apt to involve another entire lobe or large section of a lobe.

An interlobar collection of pus, following a pneumonia of either the lower or the upper lobe, would be apt to show an irregular extension of the dulness at some point beyond the line of the oblique fissure, usually about the middle of its course, but differing in outline from that caused by the gradual formation of an empyema on the one hand or of a spreading pneumonia on the other.

In thoracentesis a matter of practical importance is the selection of the point for exploratory tapping of large collections of fluid in the pleural cavity. The object in such a procedure is to penetrate the chest wall at a point where the lung has been pushed away so far that it will not interfere with the suction in the exploratory needle. When tapping is done with the vacuum apparatus for the actual withdrawal of fluid, or when the surgeon is seeking to establish permanent drainage, the lowest point of the cavity consistent with safety evidently is the optimum point of attack. With reference chiefly to adults the choice of the majority of authors is the sixth, seventh or eighth interspace between the midaxillary and postaxillary lines. In tapping more or less localized collections the area of greatest dulness usually is selected, for obvious reasons. It were well, however, to confirm such

15. Dietlen, Hans: Ergebn. d. inn. Med. u. Kinderh., 1913, xii, 197.

choice by the evidence gained from stereoscopic plates and from the fluoroscope.

The lower part of the pleural sac (Fig. 2), that which lies between the chest wall and the diaphragm, is known as the costophrenic sinus, or complemental space of Burns. It lies below the lower level of the lung during quiet respiration and extends as far down as the tenth rib in the midaxillary line (Piersol[14]). Its function is to afford space for the expansion caused by deep inspiration, but even with the most extreme effort, in an adult, it is doubtful whether the lung ever expands sufficiently to completely fill the space.

In infants and young children this costophrenic sinus is subject to even less expansion than it is in adults, owing to the less vigorous inspiratory excursions of the former. This applies with special force to the right side, where the relatively large size of the liver causes the diaphragm to rise more abruptly to its dome. During quiet respiration, therefore, the lower limits of the pulmonary lobes form the

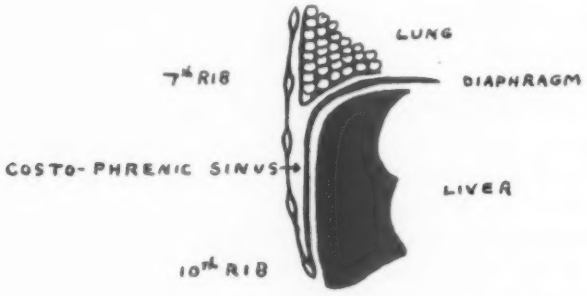

Fig. 2.—Diagrammatic sketch of the relation of liver and lung to the costophrenic sinus, in a frontal section made in the midaxillary line.

practical landmark of the lower limit of the pleural cavity, and the two layers of the pleura which line the costophrenic sinus lie almost in apposition.

When an empyema is present, this space is filled with pus, or, conceivably, may contain more fibrin than pus. While in the latter case the exploring needle might fail to reveal any fluid, the possibility of crossing the space and entering the diaphragm, and thus having a dry tap, is obvious. The serious consequences of traversing the diaphragm likewise are obvious, as on the right side the liver lies directly adjacent to the costophrenic sinus, separated from it only by the diaphragm, the parietal pleura and the peritoneum.

On the left side we found the upper border of the spleen, in the midthoracic line, lying opposite the ninth rib, or the interspace above or below it. In none of the bodies studied was the spleen greatly enlarged. Puncture of the spleen from a careless tapping through the

costophrenic sinus on the left side would be as liable to happen as would puncture of the liver on the right side. Increase in the size of the spleen naturally would increase the danger. A distended stomach also would lie directly underneath the costophrenic sinus, but unless the distention were extreme, would not lend itself easily to puncture.

We may say, therefore, that while the lowest level of the lung marks approximately the lowest level of the pleural cavity during quiet respiration, the optimum point for tapping the chest in infancy or early childhood should be somewhat higher than this level, in the interest of safe and conservative treatment.

According to Piersol[16] the lower border of the right lung in adults reaches the eighth rib in the axillary line, and the eleventh rib, or a little higher at the spine. The lowest portion of the lung is in the axillary line or a little posterior to it, but the line thence to the spine is nearly horizontal. On the left side the course is practically the same, although the left lung may descend a trifle lower at the side. Posteriorly, the lower borders are very symmetrical. Piersol further draws attention to the variations found in different types of chests, which will be referred to later.

In locating the lowest level of the edge of the lung in infants, we have selected two lines, (1) the midthoracic line and (2) a line dropped vertically from the apex of the angle of the scapula "at rest." This point is readily palpated in the living child. The results were as follows:

The lower edge of the lung in the midthoracic line on the right side was at the seventh rib or seventh interspace (7 cases), eighth rib (7 cases); on the left side it was at the seventh rib (4 cases), eighth rib or eighth interspace (9 cases), ninth rib (1 case). In the line of the angle of the scapula the lower edge was at the ninth rib or ninth interspace (12 cases), at the tenth rib (2 cases); on the left side the lower edge was at the ninth rib or ninth interspace (9 cases), tenth rib or lower (5 cases).

As would be expected, these results show that the lungs in infancy do not, on the average, reach quite so low as in adult life and corroborate Piersol's statement that the left lung reaches slightly lower than the right. We find, therefore, that the extreme lower edge of the lung, measured in the midthoracic line, in many infants does not extend below the seventh rib, while in the line of the angle of the scapula, in an even larger proportion of cases, the lowermost limit is the ninth rib.

The size of the liver, however, had less effect than would be supposed, since in the five cases which showed the greatest enlargement

16. Piersol, George A.: Human Anatomy, J. B. Lippincott Co., Philadelphia, 1907, p. 1855.

of this organ the lowest point of the lung in the midthoracic line reached the eighth rib. Although in the case with the smallest liver the lowest level was recorded as being at the eighth interspace, in two others with livers somewhat below the average size, the lowest levels were found to be the seventh rib and the seventh interspace, respectively.

An obvious objection which might be raised to the practical value of these figures fixing the lowest level of the lungs is found in the age of the specimens we examined, for the reason that empyema and pleurisy are comparatively rare in those under 1 year of age. We found, however, that in the oldest infant, one of 15 months, the level in the right side was at the seventh rib in the midthoracic line, while the lowest level, the eighth interspace, was found in an infant of only 3 months. Excluding these, the average age in five cases, in which the lower margin of the lung was at the seventh rib, was 3½ months, and in five others with the lower margin at the eighth rib, the average age was 3 months.

Since the costal, sternal and spinal diaphragmatic origins are fixed, and the development of the lungs and chest, as the child passes from infancy to adult life, is not only gradual, but also slow, it is probable that many children throughout the first decade will show the lowest level of the lung during quiet respiration at a point approximately as high as that found in some of these infants. The effect of deep inspiration at any age on the lower border of the lung of course is obvious.

In this connection we find that Piersol[17] states that the relation of the lower border of the lungs to the ribs is rendered very inconstant by the varying inclination of the ribs in chests of different sizes and shapes. We investigated this point in our studies, but we could not determine any definite relationship between the obliquity of the ribs and the lower border of the lungs. The examination of our specimens gave the following results:

When the angle of obliquity of the ribs in the midthoracic line was from 55 to 60 degrees, the lower border of the lung in the midthoracic line was at the seventh rib or seventh interspace in four cases, and at the eighth rib in two cases. When the angle was from 60 to 70 degrees the lower border of the lungs at the midthoracic lines was at the seventh interspace in one case, at the eighth rib or eighth interspace in three cases. When the angle of the ribs was from 70 to 80 degrees, the lower border of the lungs in the midthoracic line was at the seventh rib or seventh interspace in two cases and at the eighth rib or eighth interspace in two cases.

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17. Piersol, George A.: Human Anatomy, J. B. Lippincott Co., Philadelphia, 1907, p. 1856.

Although these figures indicate a tendency for the higher level of the lung to be found with the more vertical rib (55 to 60 degrees), the exceptions are too numerous to prove any definite relationship.

It would seem that the lowest point for tapping with absolute safety, therefore, would be the fifth or possibly the sixth interspace in the midthoracic line and the seventh or possibly the eighth interspace in the line of the angle of the scapula. In the authors' clinical experience it might be said that the sixth or seventh interspace in the postaxillary line (which lies between the line dropped from the angle of the scapula and the midthoracic line) is the optimum point of attack.

CONCLUSIONS

1. The fissures of the lung in infancy show practically the same relation to the bony framework of the chest as in adults.

2. The origin, course and termination of the fissures vary greatly in different individuals.

3. The variations apparently do not depend on any of the anatomic characteristics of the chest and cannot be predicted therefrom.

4. The lower level of the lungs in infants and probably in young children does not extend quite so low as in adults.

5. For this reason, and owing to the anatomic characteristics of the bases of the pleural cavities in early life, great care should be exercised to avoid damage to the diaphragm in performing thoracentesis.

6. The sixth interspace in the midthoracic line and the seventh or possibly the eighth interspace in the line of the angle of the scapula (at rest) represents the lowest limits of absolute safety for thoracentesis in early life.

FRESH AIR IN PEDIATRIC PRACTICE*

ROWLAND GODFREY FREEMAN, M.D.
NEW YORK

There is an agent of wonderful power and value to the pediatrician, the use and action of which is little appreciated even by the more prominent in the pediatric community. It has, therefore, seemed to me worth while to speak of its method of action and of its application to the uses of the pediatrician. I refer to fresh air.

By fresh air as a therapeutic agent I mean moving and cool outdoor air. The still air of the hot, humid dogdays of summer is little better than that of the crowded, hot room in winter. Fresh, moving, cool, outdoor air stimulates the appetite, induces quiet sleep, brings color to the cheeks, and increases the resistance of the organism to infection.

In seeking an explanation of the action of fresh air on the human body we find the claim that fresh, cold air raises materially the blood pressure. This claim, however, has not been confirmed by subsequent investigation, and we seem driven to the position that the favorable action of fresh air on the organism is due to the absence of the deteriorating effects of closed rooms. In fresh air the body has the advantage of normal conditions, while any modification of these, from a room with the windows partially open to a closed room, or to the conditions in the Black Hole of Calcutta, furnishes increasingly serious results from air stagnation.

The idea that air which had been breathed by other people was unhealthful probably arose from the unpleasant odor of closed and crowded rooms, and from symptoms elicited by extremes of this sort. The symptoms produced by closed places are depression, headache, dizziness, nausea, perspiration, thirst and difficult breathing. The elements producing these results were supposed to be a diminution of the oxygen and an increase of the carbon dioxid, with the possible appearance in such an atmosphere of a really poisonous product from the expired air. Experiments, however, have for the most part discredited this theory. The amount of oxygen in crowded, closed rooms is not depleted to a danger point, nor is the amount of carbon dioxid increased to such a point. Efforts to find a poisonous element in such air have

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been made from time to time with negative results. In 1911 Rosenau and Amoss[1] carried out a series of experiments on guinea-pigs to demonstrate this, but subsequent work by Weisman failed to confirm their results, and Lee[2] states "that it may be regarded as finally settled that expired air contains no volatile, toxic, organic constituent."

In 1883 Hermans[3] of the Hygienic Institute in Amsterdam became convinced of the error of the oxygen and carbon dioxid theory, and found that his own temperature was raised in crowded assemblages from three-fifths to more than a degree Fahrenheit; he concluded that the discomfort of crowded places was due to inability of the body to cool itself in a hot, moist atmosphere.

Flugge's[4] experiments in 1905 really showed the condition that produced the symptoms elicited by enclosure. He put a subject in a closed box until symptoms appeared and then allowed him to breathe, through a tube, air from the outside, without any relief of his symptoms. Another subject, outside the box, on breathing the air from the inside had no symptoms. This showed that the discomfort of a closed place has nothing to do with the air we breathe, but with conditions of the skin brought about by such confinement. We suffer in crowded, unventilated places through lack of function of the skin, rather than through respiratory embarrassment. Recent experiments have demonstrated more directly the nature of this embarrassment, for if an electric fan is placed in the corner of the box and air in the box is put in motion the symptoms disappear immediately.

These symptoms then are due to stagnant, hot, moist air surrounding the body. They may be produced in such an enclosed experimental box; they may be produced in an auditorium, overheated and crowded with people, or in a tightly closed living-room with few people, but no moving air, and such symptoms will be accentuated in people wearing heavy, impervious clothing that prevents access of moving air to the skin. It is evident, then, that we should wear as little clothing as is consistent with comfort. Most of us could wear much less clothing than we are accustomed to wearing.

The result of these elaborate observations is in brief that fresh air is good, not because it supplies oxygen, not because it is not overloaded with carbon dioxid, not because it contains no poisonous element, but because it allows the body to exist under such circumstances that it can control its moisture and temperature.

In the application of these newly developed facts to our daily work in pediatrics we have to combat the traditional fear of drafts and the

1. Rosenau and Amoss: Jour. Med. Research, 1911, xxv, 35.
2. Lee: Jour. Am. Med. Assn., 1914, lxiii, 1625.
3. Hermans: Arch. f. Hyg., 1883, L, 1.
4. Flugge: Ztschr. f. Hyg., 1905, xlix, 363.

habit of many people of living in close, hot rooms. It is only on account of the general acceptance of the brilliant results obtained in certain diseases, notably pneumonia and tuberculosis, by the use of fresh air, that we are able oftentimes to obtain the fresh air for our children which they need for the preservation of health and their proper development. Equally brilliant results possible in other diseases are not known by many physicians.

I believe that the cold air of winter is much more stimulating and produces better results in children than the mild air of spring and autumn.

Dr. Ellsworth Huntington of Yale,[5] in a very careful and interesting study of the influence of climate on civilization, concludes that spring and autumn are the periods of greatest mental and physical activity. This is contrary to the inferences I have drawn from my experiences with the influence of outdoor air and I find that his results are obtained from activities which are housed during the winter, that is to say, college students, factory workers, etc. These people are all housed in winter and live in warm air and thus are little affected by the tonic action of cold outdoor air. In spring and autumn, on the other hand, the windows are opened. If he had used outdoor occupations for his studies I believe his results would have been different.

The best results from fresh air are obtained by keeping the children out of doors day and night, and we have no healthier children than those who, having suffered in early life from tuberculous infection, have been kept for years out of doors. These children are not subject to colds which others have during the winters; they have good color, good appetite and a normal, robust development.

Many of our pediatricians have confused fresh air with cold air, and one may see in hospitals cold wards and warm wards. Such cold wards are probably better than warm wards, but they lack altogether the free, moving air which is the great advantage of the outdoor ward, aud often the sunshine, which we know to be one of the most potent of our therapeutic agents.

Outdoor sleeping porches enclosed on three sides and roofed, but open to the south, furnish the best fresh air at night, while in the daytime balconies and rooms without heat and windows wide open supply the air we need.

It is evidently not enough, however, that we should have this fresh air, but we should also look to the clothing to see that our children are not sealed in with heavy, impervious covering so that the skin is unable to rid itself of the heat and moisture.

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5. Huntington, Ellsworth: Civilization and Climate, Yale University Press, 1915.

The experiments I have reviewed show us how it might not be impossible for a child out of doors, in cold weather, to be embarrassed by such clothing.

Where it is impossible to obtain such complete outdoor exposure, the best substitute in cold weather has seemed to me to be in rooms with cheesecloth screens in the windows. Such cheesecloth screens should cover all the space available when the windows are opened. They allow a moderate access of air without the presence of the much-feared drafts. The objection is that they allow less sunlight than glass, but this disadvantage is more than counteracted by the quality of the air of the room.

Other methods of ventilation consist in patent ventilators put under the lower sash. These, however, allow more movement in the air of the room, but less open space, and some of them furnish no filtration of the dust from the air.

The greatest blight on the fresh air movement of the day is the vogue of elaborate, mechanical ventilating systems, which obtain the air from the basement, pump it through flues into and out of the rooms. Such systems are very expensive, occupy a great deal of space, and while the air furnished may be sufficient for the preservation of life, it often lacks the freshness and invigorating character of the air obtained from open windows or through cheesecloth screens. The worst phase of the current ventilating systems is when they prohibit the opening of the windows. Under intelligent operation they may prove efficient. Closed rooms favor the conveyance of disease from one person to another, and our ventilating systems make practically no provision for carrying off germ-laden dust. There is little conveyance of disease in outdoor school classes or outdoor hospital wards.

The danger of housing is not alone in the bad air of a closed room, but to a susceptible organism it consists also in the sudden change from a temperature of 70 or 80 in a dry room all night to a temperature perhaps of zero, with a cold north wind blowing out of doors, then to a heated auditorium, with a saturated, moist temperature of 80, and again out into zero weather. It is only during the existence of marked changes of temperature between indoors and outdoors that epidemics of colds exist, for during the summer we have practically an immunity to colds and colds occur only when our houses are closed.

In New York our epidemics of colds usually begin in November and December, while in New Hampshire, where I often spend my summers, cold weather inducing people to heat their houses often occurs in September, and it is not uncommon for the neighboring town to have a severe epidemic of colds at such a time. In the summer

time the air is less invigorating, but there is the advantage of less sudden change from cold to hot or from dry to moist air.

Much is heard of the superiority of country air over city air, but recent investigations have shown but little difference in its chemical composition, and if the children are kept in the open air, as they should be in the city, they are much healthier than most country children.

Night outdoor air, while it is not so good as day air because it lacks the beneficent effect of sunlight, is far superior to night air in the house, and the advantage of fresh air treatment is reduced by half if children are housed at night.

A statement as to the diseases to which fresh air treatment is applicable is much more difficult than a statement of the diseases to which it is not applicable. I will, therefore, first enumerate the conditions to which fresh, cold air has not been demonstrated as practicable.

Premature infants who show a subnormal temperature in cool air should be kept at an air temperature that will preserve the normal body temperature. This warm air, however, must be a freely moving warm air, rather than the dead air found at the bottom of a box. On this account, for such babies, a properly constructed incubator is far superior to the substitutes that are often adopted.

I am not sure whether such cold, fresh-air treatment is applicable to children with kidney lesions or with severe heart lesions, although I have seen children with severe endocarditis who preferred to be out of doors in winter rather than in closed rooms, and I have seen no injury from such exposure.

The most important application of this fresh-air treatment is to build up the vitality and resistance to disease of frail children, and in this sphere, I think, it shows perhaps its most brilliant results. This is true in early life, for children with marasmus and malnutrition who are impossible to feed so as to obtain an increase in weight will often gain promptly, with no change in food, on being kept out of doors or in front of an open window in winter. I have seen many children who have reached that stage of malnutrition that usually precedes death, with a dusky hue of the face, react to such treatment and recover entirely.

Another condition of early life, rachitis, is, I believe, entirely a disease of housing. It exists, not in tropical climates where people live out of doors, but in colder climates where people house themselves in winter. The symptoms develop in winter only and the severe cases that we see are entirely confined to the children of races that have been accustomed to warm climates where the families do not house themselves in winter. This susceptibility is not confined to human beings, but exists equally in the lower animals. Animals brought here

from tropical climates develop a disease that is in many respects similar to our rachitis and which is known in zoological gardens as cage-disease, and this is the fatal disease that all zoological gardens have to combat and from which none will be free until greater care is taken to supply these animals with fresh, cool, moving air in winter. On account of this susceptibility Italians and colored people and other people accustomed to tropical climates should be warned that they must give their children fresh air in winter if they would have them survive and develop properly.

In all the acute infectious diseases I think there is now a general acceptance of the advantage of fresh air, excepting perhaps in measles and scarlet fever. In tuberculosis there is now no question of its advantage. In pneumonia the results from this treatment have exceeded those from any other method of treatment, including specific treatment with serums and vaccines. There are still many men, however, who, while allowing that fresh air is beneficial in an inflammation of the lung, doubt its efficacy in bronchitis, pharyngitis, laryngitis, rhinitis and otitis. The only reason why such men doubt its efficacy is because they have not sufficiently tried it. I have used it in all these conditions and I believe that it is of direct benefit and that it diminishes materially the mortality.

I have convinced myself also that measles and scarlet fever are no exceptions to the rule, and I have put patients during a severe measles epidemic complicating scarlet fever out of doors in winter without a single fatality, although some of the scarlet fever patients were exceedingly sick when the change was made. The change to outdoor air seemed to help them all. Among these cases was one of scarlet fever in which from a breaking down of the lymph nodes there was a large ulcer extending from one side of the neck to the other. Under the influence of the cold air the neck healed with a linear scar. It is equally unnecessary in measles to darken the room. We should let children with this disease have all the sunlight that their eyes will bear, as well as cold, fresh air.

In connection with the healing of this surgical condition complicating scarlet fever, the statements made by H. S. Soutter[6] are of interest. He says that they received wounds that were soiled by earth, manure and fragments of cloth covered with mud and that there was one way by which all such infections may be defeated, by plenty of fresh air or oxygen. In several cases, in which the wounds were so horribly foul that it was impossible to tolerate them in the wards, the patient was put in the open air with the wounds covered only by a thin piece of gauze. The results were almost magical. The wounds lost their

6. Soutter, H. S.: A Surgeon in Belgium. 1915.

odor and began to look clean, while the patient lost all signs of the poisoning which had been so marked before. He thinks this treatment may account for the fact that they had no case of tetanus.

J. W. Markoe states his experience with open-air treatment in sepsis at the Lying-In Hospital in New York. Of fifty-seven patients treated indoors, two left the hospital and fifty-five died; that is, all the patients who remained in the hospital died; while of fifty-three patients who were put out of doors, twenty-two, or 41 per cent., recovered.

The common colds of winter are favorably influenced by cold, fresh air, as are other forms of infections.

In diseases characterized by abnormal conditions in the blood, such as simple anemia, von Jaksch's anemia and leukemia, I believe there is a large field of usefulness for this treatment, and I have myself, in a very limited number of cases, obtained quite brilliant results.[7] These observations must, however, be confirmed by many more before one can consider this claim as proved. Markoe, however, states that patients with anemia from severe hemorrhage are greatly benefited by fresh air.

Before closing, it would seem that some explanation is due as to why, if all these statements are true, children are still housed and many adults have a panic if a breath of cold air strikes the back of their necks or their bald heads, while children who are brought up without fear of cold enjoy it wherever it strikes.

The supposed production of catarrhal inflammations in adults by exposure to cold air, if it really exists, exists only on account of suggestion. These people have been brought up to such a fear of fresh air that every infection of the upper air passages to which they succumb they attribute to this health-giving influence. It is sincerely to be hoped that many of the coming generation may be brought up under different ideas and may be less dependent on hot, offensive, stagnant air for the supposed comforts of life.

There is evidence enough to show that many diseases are favorably influenced by this simple and safe measure. Why do you not use it? Some are afaid, some will not take the trouble. Many children are allowed to become sick from housing and children may be seen dying in closed wards of many of our best hospitals who might have been saved had they been put out of doors.

211 West Fifty-Seventh Street.

7. Freeman, R. G.: Am. Jour. Med. Sc., 1916, cli, 1.

CLINICAL DEPARTMENT

A CASE OF INFANTILISM DUE TO HYPOPITUITARISM *

H. CLIMENKO, M.D.
Attending Physician to the Central and Neurological Hospital
NEW YORK

For centuries the pituitary gland has been one of the most mysterious structures of the human body; some have considered it the seat of the soul, others have looked on it as a rudimentary organ, while most physiologists have confessed total ignorance as to its functions in the human economy. The discovery in 1886 by Pierre Marie of the association of changes in the structure of this gland with acromegaly has stimulated further and detailed studies of the physiology of the epiphysis, and it was found that it is endowed with a number of vital functions, such as regulation of blood pressure, contraction of smooth muscle, particularly the uterus; the sexual functions, and the growth of connective tissue and of the skeleton. Moreover, it was found that the pituitary has a great influence on the mentality of its possessor.

Among the pioneers in the study of the physiology of the pituitary may be mentioned Frankel-Hochwart, Froelich, Cushing, Vincent, Cyon, and others. But it appears that there remains much yet to be learned about this organ.

One of the most interesting phases of the study of the pituitary function is its relation to infantilism, a syndrome which has been obscure for centuries. E. J. Mullally,[1] A. W. Hewlett,[2] George E. Rennie[3] have reported cases of infantilism with pathologic changes in the pituitary gland found at necropsy. The case here reported appears to belong to this class, because the symptoms pointed undoubtedly to the infantilism being due to perversion of function of the pituitary.

The patient, Y. K., a girl 12 years old, was born in this country of Russian-Jewish parentage. The mother gives a history of two miscarriages and the fact that she is markedly prognathous, her jaws protruding like a muzzle, may be suggestive. On the father's side there is a history of tuberculosis. It is also noteworthy that the mother, sisters and brothers are of unusually large size, especially when we bear in mind that they are Jews who are

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* From the Neurological Division of the Mount Sinai Hospital Dispensary.
* Case presented before the New York Neurological Society, June 6, 1916.
1. Mullally, E. J.: Arch. Int. Med., 1913, xi, 523.
2. Hewlett, A. W.: Arch. Int. Med., 1912, ix, 32.
3. Rennie, G. E.: Brit. Med. Jour., 1912, i, 1355.

ethnically of low stature. Repeated Wassermann examinations of the blood from the mother and child proved negative. A von Pirquet inoculation also gave negative results. No forceps were used during the delivery of the patient. She was breast fed and, according to the mother's statement, began to walk at 2 years of age. At the age of 18 months the child suffered from whooping cough.

When the infant was 2 years of age the mother was rather alarmed at its stunted growth and applied to a hospital for treatment. She tells me that the powders given to the child were quite effective at the time, but unfortunately I am unable to ascertain the nature of the medication.

Fig. 1.—Photograph of patient suffering from hypopituitary infantilism.

When the child was brought to me on March 5, 1916, the mother pleaded for relief, stating that although the child is rather cunning and shrewd, she is backward in her studies at school. Moreover, the mother complains that the child is not promoted in the school because of her stunted size. No history of frequent micturition or of enuresis is obtainable.

The patient appears the size of a child of 4. The face and mucous membranes are pale, the hair fine and scanty. The subcutaneous adipose tissue is rather excessive, but the skin can be raised in folds. The abdomen is protruding, and the umbilicus is situated lower down as is the case in the infantile type.

Examination of the cranial nerves gives negative results, excepting the optic disks, which are pale, but there is no change in the visual fields and

the acuteness of vision is normal. The reflexes are normal, and no sensory changes could be discovered. The neck is well formed, the thyroid is not palpable. Nothing of significance could be ascertained by an examination of the lungs. The urinary findings are negative.

The following table gives some anthropometric data about the little patient:

Weight	46	lb.
Height	98	cm.
Lower extremities	46	cm.
From anterosuperior spine to upper border of the patella	25	cm.
From upper border of patella to internal malleolus	21	cm.
Upper arm (from acromion to tip of olecranon)	17.5	cm.
Lower arm (from olecranon to styloid process of the radius)	14	cm.
Distance between anterosuperior spines	24	cm.
Circumference of the chest	59	cm.
Circumference of the abdomen	63	cm.

No striking asymmetries have been found.

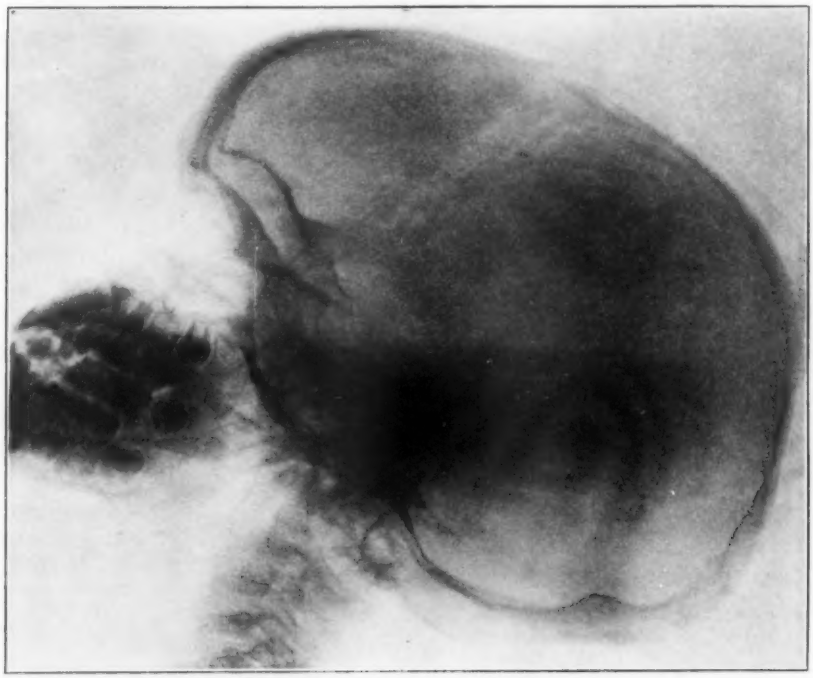

Fig. 2.—Roentgenogram of the skull of the patient, showing a sella turcica encroached on by bony projections from its anterior and posterior clinoid processes, as well as from the base. Note the thinness of the frontal and parietal bones.

The radiographic findings (Fig. 2) show an encroachment on the cavity of the sella turcica by bony projections from the anterior as well as the posterior clinoid processes, also projections from the base of the sella. The entire skull is very thin and the frontal and parietal bones show a peculiar mottling, while the anterior fontanel is not completely closed. There is also lack of ossification of the long bones (Fig. 3).

Several times glucose was given to the patient while on a fasting stomach running up from 50 to 200 gm., and no sugar was found in the urine at any

time, thus showing the high sugar tolerance. Five drops of a 1:1,000 solution of epinephrin were given hypodermically, and no sugar was obtained in the urine.

Examination of the blood shows the following results: Hemoglobin, 60 per cent.; erythrocytes, 3,500,000; white blood cells, 6,000. No abnormal cells, and the cell count was negative. The results of a lumbar puncture were negative.

The Binet-Simon test shows that the child has the intelligence of one 7 years old. She is, however, very poor in arithmetic. She can count correctly, but

Fig. 3.—Roentgenogram of hands and forearms of patient, showing defective ossification.

finds difficulty in counting backward. Addition and subtraction of small and simple numbers she does not perform with ease or correctly most of the time.

For about a month the child was fed on thyroid extract. Various doses were given, beginning with $\frac{1}{16}$ of a grain to 1 grain without any noticeable changes. Then pituitary extract was administered (the entire desiccated gland). At the end of a month an increase in weight of half a pound was found. At

the end of three months, one pound increase, the child now weighing 47 pounds. No increase in the size of the child was as yet noticed.

Considering the conclusions arrived at by E. A. Schaffer,[4] to the effect that the anterior lobe is probably responsible for the growth of the body, we may rightly call into question the old theories of Loraine, Brisaud, and Sanct de Sanctis concerning the nature and origin of infantilism. Loraine thought that all forms of this condition were due to infections. This is, of course, a vague term and can easily be dismissed at the present state of our knowledge of the physiology and the pathology of the pituitary. The thyroid was held responsible for infantilism by Brissaud, while Sanct de Sanctis thought the condition due to pluriglandular disturbances.

The roentgenogram, as well as the clinical findings of this case, point to the pituitary being primarily responsible for the stunted growth. Whatever thyroid involvement there is may be due to the usual response of this gland to changes in the pituitary.

SUMMARY

Our patient presents undoubtedly the syndrome of infantilism caused by a hypofunction of the pituitary. Our reasons are the following:

The bony projections in the sella turcica must of necessity interfere with the function of the pituitary by the pressure it exerts on this gland. On the other hand, should we assume that the bony projections are of embryologic origin, it is clear that, even so, the pituitary was hindered in its development.

The high sugar tolerance, both to glucose and to epinephrin, the lack of ossification of the long bones, the anemia, the absence of distinct myxedematous symptoms and signs, lead us by exclusion to the pituitary for the cause of the stunted growth.

This case, with the above mentioned cases, tends to prove that infantilism is in all probabilities due to hypofunction of the pituitary gland.

252 East Broadway.

4. Schaffer, E. A.: Proc. Roy. Soc. Med., lxxxi, 442.
lxxxi, 442.

TRANSIENT ABDOMINAL TUMOR IN A CHILD OF FIVE YEARS WITH REDUNDANT COLON *

GEORGE N. ACKER, M.D.
AND
EDGAR P. COPELAND, M.D.
WASHINGTON, D. C.

The complaint in this case was the periodic occurrence of abdominal tumor. The patient was the only child of young and healthy parents, though the mother might be said to be of a nervous temperament. The birth weight was 11¾ pounds and the labor was tedious, though a head presentation, delivery being finally effected, after forty-eight hours, by instrumentation. No difficulty was experienced, however, in establishing respiration, and the infant was normally nourished until two days after birth, when a promising lactation for some reason failed. After this the child ran the gauntlet of proprietary foods, including Eskay's, Mellin's and finally Horlick's malted milk, which was continued well into the second year. He sat up at five months, began the eruption of teeth at eight months and walked at nineteen months. With the exception of frequent attacks of rhinitis, to correct which an adenoid operation was performed at the age of 2½ years, the patient escaped all of the diseases peculiar to childhood, progressing in a fairly normal manner to the age of 3½ years.

In December, 1914, approximately a year before my first examination, the patient became suddenly ill in the night, with an attack characterized by extreme nausea and severe vomiting and the appearance of a rounded tumor in the hypogastrium, simulating a distended bladder. The vomiting, to judge from the description, was simply bile-stained gastric juice and at no time stercoraceous. The tumor was elastic, but not especially tender to touch. There was no history of previous disturbance in the regularity of the bowel, as to constipation or diarrhea. Fever was not present.

The physician called at the time had evidently made a diagnosis of intussusception and had completed plans for an immediate removal to the hospital for operation. Returning a few hours later for the patient, he had been, as could be well imagined, much surprised to find that the mass had spontaneously disappeared and the patient recovered.

After this initial appearance these attacks had recurred at varying intervals, seldom less than three weeks and on several occasions as long as six weeks. They had varied in the severity of associated symptoms and likewise in duration, seldom, however, lasting over two days. The tumor had invariably appeared first over the region of the bladder, sometimes larger, sometimes smaller, moved about the abdomen spontaneously and finally disappeared. Its appearance had always been associated with nausea and vomiting, and its disappearance with a pronounced paroxysm of abdominal pain. Following the first attack there had been some tendency to constipation, but the bowels had been kept freely open by the daily employment of mineral oil by mouth. The diet had been well regulated and in the intervals the patient had quickly recovered lost weight. Ordinarily the child was quite normal, played actively and appeared in good health. No prodromal symptoms had been observed.

At the time of my first examination, I found the patient in bed lying on his back, thighs partially flexed. The attack was several hours old and there was

* Submitted for publication May 29, 1916.
* Read at the Twenty-Eighth Annual Meeting of the American Pediatric Society, held at Washington, D. C., May 8-10, 1916.

still some nausea. Presenting in the hypogastrium was a smooth tumor about the size of an orange, elastic, but not tender to touch, and dull on percussion. It was palpable by rectal examination and suggested strongly a distended bladder. The mass was, however, freely movable, it being possible, without undue force, to manipulate it about the entire abdomen. There was a fairly well-pronounced beading of the ribs. The pulse was rapid, but regular. The temperature normal. A leukocyte count gave 11,500. The von Pirquet and Wassermann tests were negative. No further significant facts were observed.

Fig. 1.—Roentgenogram taken tenth minute after bismuth flow commenced.

Under restricted feeding and large enemas slowly administered, the mass spontaneously disappeared. An examination of the abdomen subsequently was absolutely negative.

Through the courteous cooperation of Dr. J. H. Selby, a very thorough Roentgen study of the case was made between and during attacks. In this connection I believe that I cannot do better than read Dr. Selby's own report. Examination was made March 5, 1915:

"Findings: Original plates, both back down and face down before the bismuth enema, were negative. Twelve ounces of suspended bismuth subcarbonate were injected by gravity and the visualized filling observed. Plate records were made at the second, fourth, seventh, tenth and thirtieth minute after the flow commenced. The ampulla filled normally, the bismuth column passing vertically, almost in a straight line, to the level of the second lumbar, slightly to the left of the midline, where it turned sharply downward a distance of six inches to the left iliac fossa, at which point it turned sharply on itself

Fig. 2.—Roentgenogram taken at twenty-fourth hour after bismuth enema.

passing upward to the diaphragm, when it again turned on itself and descended at a posterior level for three inches before crossing as the transverse colon at the second lumbar. The hepatic flexure is on a level with the transverse colon. The cecum occupies a position one inch above the right iliac crest. The bismuth column reached the cecum in six minutes. *By manipulation the redundant sigmoid or distal portion of the colon can be made to swing over toward the right side and back to the left at will.*

"At the twenty-fourth hour there is considerable bismuth residue in almost the entire colon. The aforesaid redundant colon is distinctly outlined.

"Examination made March 27, 1915:

"Findings: A tumor mass the size of a large orange is palpated in the right upper quadrant. The bismuth injection showed the mass to be redundant descending colon. Visualized palpation reduced the tumor."

CONCLUSIONS

The clinical history, in the light of the Roentgen-ray findings, would seem to justify the assumption that the phantom tumor was the result of a temporary kinking of the redundant colon (or sigmoid), incident to its displacement to the right, which was followed by either fecal or gaseous distention in the loop. When the loop filled itself to a certain point, it swung gradually to the left and automatically unkinked itself, with a disappearance of the tumor mass.

CONGENITAL OBLITERATION OF THE AORTA

WITH REPORT OF A CASE *

HARRY GAUSS, S.M., M.D.
CHICAGO

Congenital obliteration of the aorta occurs in two main forms: closure at the orifice, so-called atresia ostii aortae; and at the isthmus, referred to as coarctation.

These two forms present different clinical aspects. Coarctation is of greater frequency. Abbott[1] states that over 200 cases have been reported. This lesion occurs in that part of the aorta lying between the left subclavian artery and the insertion of the ductus arteriosus. Patients having this anomaly may reach adult life. Recently LeCount[2] demonstrated a specimen removed from the body of a man 42 years old. The greatest age reached occurred in an instance recorded by Reynaud, cited by Hochsinger,[3] in which the patient was 92 years old. The clinical signs, as summarized by Hirschfelder,[4] are difference in the size and quality of the pulse in the upper and lower extremities; difference in the blood pressure in the upper and lower extremities; a low murmur over the arterial trunks, frequently at the angle of the left scapula, and the presence of large tortuous mammary, thoracic and scapular arteries. As to the etiology, Martens[5] gives three causes: the influence of traction exerted on the isthmus by the ductus arteriosus, especially during its involution; changes in the vessel wall analogous to an endarteritis fibrosa, first suggested by Thoma; a congenital malformation of the vessel, the latter view being supported by the frequent concomitant occurrence of other congenital abnormalities, which have been noted in 38 per cent. of the recorded cases.

Atresia ostii aortae is less common. This lesion occurs at the orifice of the aorta. Humphry[6] cites Rauchfuss, who collected twenty-four cases. Patients with this abnormality generally do not live more than a few weeks. In an instance recorded by Bardeleben and cited by Martens, the age of 27 weeks was reached. This lesion is generally

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* Submitted for publication July 1, 1916.
* From the Department of Pathology, Michael Reese Hospital.
* Read before the Michael Reese Clinical Society, Jan. 3, 1916.

1. Abbott, Osler and McCrae: Modern Medicine, 1915, iv, 399.
2. LeCount: Tr. Chicago Path. Soc., 1914, ix, 88.
3. Hochsinger: In Pfaunder and Schlossman, Diseases of Children, iii, 474.
4. Hirschfelder: Diseases of the Heart and Aorta, 1913, p. 547.
5. Martens: Virchows Arch. f. path. Anat., 1890, cxxi, 322.
6. Humphry, Allbutt and Rolleston: System of Medicine, 1909, vi, 286.

associated with aplasia of the left ventricle and very frequently other abnormalities, which lend support to the view that it is a congenital malformation. Monkeberg[7] recognizes an inflammatory type. He records an instance of a patient four days old, born spontaneously after a normal labor. He bases his view on the results of the study of microscopic sections.

Our specimen of congenital atresia of the aorta at the orifice was encountered in a routine postmortem examination.

CASE REPORT

History.—The infant boy was born in the maternity section of the Michael Reese Hospital, Oct. 17, 1915, a patient of Dr. Braude. The mother was a primipara, who had passed through an uneventful pregnancy. Labor had been normal and delivery spontaneous. The child appeared to be well formed and presented no external abnormalities. The heart sounds were normal; no murmurs were heard, the cry was normal, the skin was clear and of good color. On the second day the child was put to the breast, and apparently nursed normally. On the third day it suddenly became cyanotic, went into convulsions, and died within a few hours.

Necropsy.—The anatomic diagnosis was: congenital atresia of the aorta at the orifice; total absence of the aortic valves; hypertrophy of the left ventricular wall; dilatation of the right ventricle; common opening of the coronary arteries; partial apneumatosis of the lungs; multiple small hemorrhages in the epicranium, pericardium, liver, kidneys, thymus and suprarenals; marked passive congestion of the liver, spleen and kidneys; uric acid infarcts in the kidneys; cloudy swelling of the liver; hydrocele; adherent umbilical cord.

The body was that of a well-nourished white infant boy, measuring 48 cm. in length and weighing about 7 pounds. Rigor mortis, algor mortis and postmortem lividity were present. The anterior and posterior fontanels were open, and the bones of the skull were movable at their sutures. The pupils were slightly dilated, equal in size, the corneas were clear, the nose was well formed. The chest and abdomen appeared normal externally. The proximal end of the umbilical cord was adherent, measured 4 cm. and was dried and atrophied. The scrotal sac was slightly enlarged and contained clear serous fluid. The costochondral junctions and the ends of the long bones appeared normal.

The body was opened by the usual longitudinal incision. The subcutaneous adipose tissue was normal in amount. The peritoneum was smooth, glistening and transparent, and there was no free fluid in the peritoneal cavity. The liver extended 5 cm. below the costal arch, the stomach was slightly distended and the appendix appeared normal.

The heart lay free in the pericardial sac, which contained the usual amount of clear serous fluid. It was slightly enlarged, measured 37 mm. from the apex to the base, and weighed 23 gm. The apex was made up of the right ventricle, which was appreciably larger than the left ventricle. The auricles and the coronary sinuses were distended with blood. Beneath the visceral and parietal pericardium there were multiple small hemorrhages. The heart was opened by two incisions into the ventricles parallel to the interventricular septum. The right ventricle was dilated and measured 40 mm. transversely and 33 mm. from its most dependent point to the pulmonary artery. The ventricular wall was 3 mm. wide. The pulmonary artery was considerably larger than the aorta; exposed and laid open, it measured 25 mm. along the line of the valve attachment. The valves of the pulmonary and those of the tricuspid opening appeared normal in structure. The left ventricle was considerably smaller than the right; it measured 10 by 11 mm. The lining endocardium was

7. Monkeberg: Deutsch. path Gesell., 1907, xi, 224.

smooth and glistening. The left ventricular wall was hypertrophied, and measured 10 mm. The mitral opening was smaller than the tricuspid opening, was made up by two valves, which did not approximate well, but which were otherwise well formed. From the ventricular cavity there was no opening into the aorta; but at the site of the aorta there was a small pinpoint retraction of the endocardium, and there was no evidence of the aortic valves. The aorta was completely closed for a distance of 2 cm.; beyond this point it dilated and reached its normal diameter at the junction of the ductus arteriosus. The closed aorta was 2 mm. wide. The coronary arteries opened into the left ventricular cavity by a common orifice near the site of the aorta close to the interventricular septum. The right auricle was slightly dilated, but normal in structure; the left auricle was smaller than the right and presented no abnormalities. The ductus arteriosus and foramen ovale were widely patent.

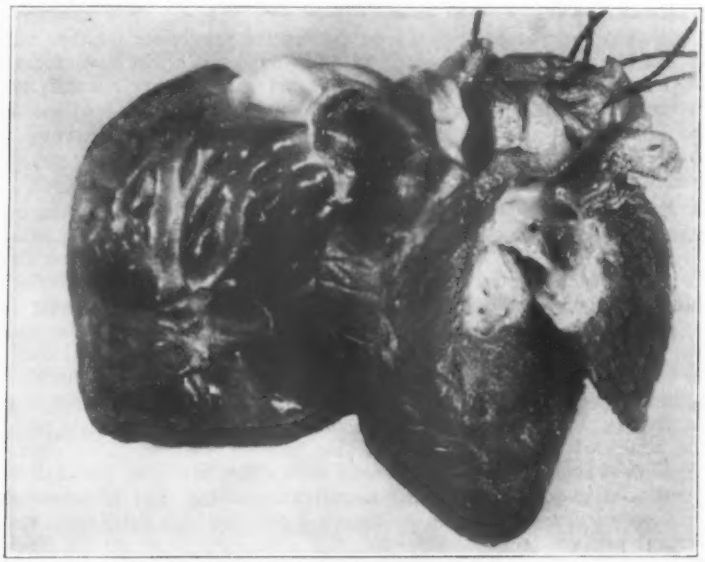

Fig. 1.—Photograph of heart, anterior surface, dissected.

The lungs lay free in the pleural cavities. The right lung was normal in size, weighed 30 gm., was pinkish red in color, and floated low in water. It was soft and friable, except a few small areas which were firm and deep red in color. Sections of these areas sank in water. From the freshly cut surface a frothy hemorrhagic fluid escaped. Beneath the pleura and in the substance of the lung there were multiple small pinpoint hemorrhages. The left lung weighed 28 gm. and also contained small hemorrhages.

The thymus was dark red in color, weighed 7 gm., measured 4 by 3 by 1 cm., and contained numerous small hemorrhages.

The liver was enlarged, was dark red in color, measured 11 by 6.5 by 5 cm., and contained numerous subcapsular hemorrhages. The lobular markings were distinct. From the freshly cut surface of the liver dark fluid blood escaped; the surface appeared dull, and the edge everted. The gallbladder contained about 3 c.c. dark green, tenacious fluid.

The spleen was normal in size, weighed 10 gm., was congested, and the cut section was dark red in color.

The right kidney contained numerous small subcapsular hemorrhages. It was normal in size, fetal lobulation was present, and it measured 4 by 3 by

2 cm. The capsule stripped easily, leaving a smooth, shiny surface. On cut section, the cortex appeared dull, the medulla was intensely congested and at the pyramids there were several yellowish-white uric acid infarcts. The left kidney presented the same alterations.

The head was opened, the brain removed and sectioned. There were multiple small hemorrhages in the epicranium, but otherwise there were no abnormalities.

As this infant lived several days extra utero, besides passing through its normal intra-uterine life, some explanation of the circulation should be forthcoming. In the normal fetal circulation (Wil-

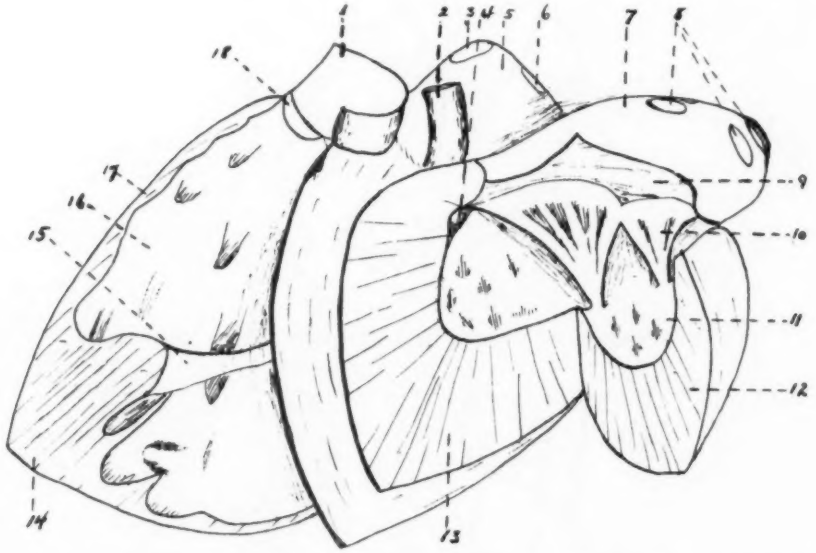

Fig. 2.—Sketch of anterior surface of the heart corresponding to photograph shown in Figure 1. 1, pulmonary artery; 2, aorta; 3, superior vena cava; 4, common opening of the coronary arteries; 5, right auricle; 6, inferior vena cava; 7, left auricle; 8, pulmonary veins; 9, mitral orifice; 10, mitral valves; 11, left ventricular cavity; 12, left ventricular wall; 13, interventricular septum; 14, apex of heart; 15, large papillary muscle of right ventricle; 16, right ventricular cavity; 17, right ventricular wall; 18, pulmonary valves.

liams[8]) the blood from the superior vena cava enters the right auricle, passes into the right ventricle, then into the pulmonary artery, where it divides, some passing into the pulmonary circulation, the greater part passing through the ductus arteriosus into the descending aorta. The blood from the inferior vena cava enters the right auricle, passes through the foramen ovale into the left auricle, then into the left ventricle which pumps it into the ascending aorta (Fig. 3). The following explanation of the circulation of this instance of closure of the aortic orifice is offered, which explains at least a possible circulation.

8. Williams: Obstetrics, New York, 1912, p. 156.

Blood from the superior vena cava followed the same course as in the normal fetal circulation. Blood from the inferior vena cava entered the right auricle, passed through the foramen ovale and entered in whole or part the left ventricle where it was forced back through the

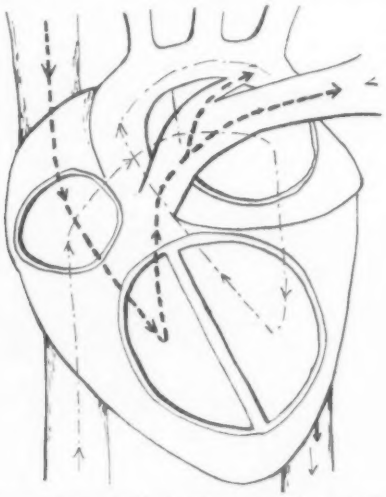

Fig. 3.—Diagram of normal fetal circulation (after Spalteholz).

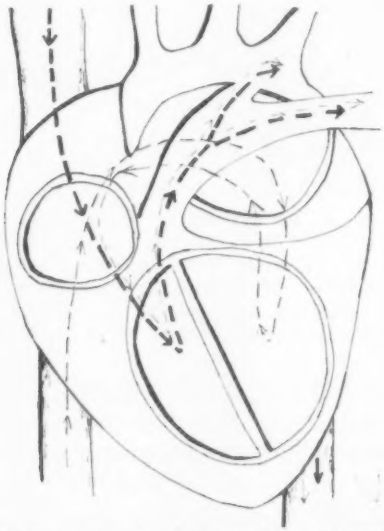

Fig. 4.—Diagram of circulation with obliterated aorta.

foramen ovale into the right auricle, where it went with the blood from the superior vena cava into the right ventricle, then into the pulmonary artery, where it divided, part going into the pulmonary circulation and the remainder into the aorta through the ductus arte-

riosus. Blood returning from the pulmonary circulation into the left auricle was forced by contractions of the left ventricle through the foramen ovale into the right auricle, where it mixed with the other blood (Fig. 4). Such a circulation could be successfully continued as long as the right ventricle was able to maintain the systemic as well as the pulmonary circulation, with death occurring when the right ventricle failed.

I wish to thank Drs. E. G. Kirk and H. Gideon Wells for their assistance.

THE USE OF CREAM AND PRECIPITATED CASEIN IN INDIGESTION WITH FERMENTATION*

JOSEPH I. GROVER, M.D.
Junior Assistant Physician to the Children's Hospital
BOSTON

A few months ago I had the opportunity of studying a very interesting feeding case in the outpatient department of the Children's Hospital. The subsequent feeding history deserves special attention.

Baby D., birth weight 8 pounds, nursed one week, was then put on various formulas of milk, water, and lime water, and sometimes Mellin's food. All the formulas were of fair proportions. She came to us on June 21, 1915, at the age of 10 weeks, weighing 7 pounds 1 ounce, with a history of projectile vomiting, hunger, and markedly excoriated buttocks. The sample stool that day was hard and yellow. She was given a trial laboratory formula of 1 fat, 4.5 lactose, 1.5 protein, limewater 50 per cent. of the milk and cream.

The accompaying table shows the date of each visit, the weight, the number of stools a day, and the formula ordered at that date. The plus signs in the column showing the number of stools indicate a diarrhea with no definite number mentioned.

Explosive vomiting followed the taking of the trial formula, and the baby passed a great deal of gas. The case was considered one of indigestion of fats, and the baby was put on a formula containing no fat, which furnished 57 calories per kilogram, and the food was boiled.

This formula did not agree any better than the previous one. The mother of her own accord gave a mixture of condensed milk and water. The baby gained 25 ounces in forty-one days on it. For the first few days it did not vomit, but then it began again as badly as ever. Everything on the whole went fairly well until four days before the next visit, when a diarrhea started, with many loose, green, slimy stools, containing streaks of blood. The case looked like an infectious diarrhea, but there was no rise of temperature. The stool was planted for gas bacillus, and the baby was given a 3 per cent. barley water, with 2 per cent. lactose.

It took the mixture well and vomited but little, yet the number and character of the stools remained the same. The gas test on the stool was positive. This test was done twice later and found negative each time. No change was made in the food. On August 7 milk of bismuth was given in 0.5 dram doses after each bowel movement.

In two more days, on the 9th, the mother reported vomiting, from ten to twelve watery stools without blood or pus, and that the baby was in rather poor condition. The buttocks were still excoriated. The "formulas" from this time to the end of August were fat free, or with small amounts of fat, contained 4 per cent. or less of total lactose, a high protein, and some lime water. The stools often showed a marked excess of soaps even on the skimmed milk.

On September 1 Dr. Sylvester, head of our outpatient clinic, was consulted, and we concluded that the lactose was responsible for the vomiting, diarrhea and excoriations, which were on that day more marked than ever.

I ordered a laboratory formula in which precipitated casein was substituted for the protein in the skimmed milk. The percentage of lactose in precipitated

* Submitted for publication April 27, 1916.
* From the outpatient department of the Children's Hospital.
* Read before the New England Pediatric Society, January, 1916.

casein is about 0.1, according to a chemist's analysis. In putting 2 per cent. precipitated casein into a formula, only 0.002 per cent. lactose would be added thereby. The cream adds about 0.12 per cent. to each 0.75 per cent. fat, as a 32 per cent. cream is always used by the laboratories. Thus, in a mixture containing 0.75 per cent. fat and 2.5 per cent. precipitated casein, there would be present about 0.13 per cent. lactose. On September 1 the baby was given a formula representing 0.75 per cent. fat, 0.12 per cent. lactose, 2.5 per cent. precipitated casein, with a little lime water. A little paregoric was given.

OUTLINE OF FEEDING HISTORY OF BABY D., THE QUANTITIES IN THE FORMULAS INDICATING PERCENTAGES

Date	Weight, Lb.	Oz.	Stools*	Fat	Lactose	Dextri-Maltose	Maltose	Skimmed Milk Protein	Precip. Casein	Lime Water†	Barley Starch	Ounces Daily
6/21	7	1	..	1	4.5	....	....	1.5	.....	50	....	24
6/25	7	..	2	0	4.5	....	....	1.2	.....	50	0.5	..
8/ 4	8	9	+	.....	2	....	....	.....	.....	....	3	32
8/ 7	8	3	+	.....	.....	....	....	.....	.....	....	....	..
8/ 9	7	14	11	0	2.75	....	....	2	.....	40	....	48
8/10	8	6	8	.....	.....	....	....	.....	.....	....	....	..
8/13	8	9	10	0	2.5	....	....	2.1	.....	40	....	..
8/16	8	6	5	0	3	....	....	2.5	.....	30	....	..
8/18	8	5	4	0.25	4	....	....	2.5	.....	30	....	..
8/23	8	5	8	0.25	3.5	....	....	2.5	.....	10	....	..
8/25	8	5	+	0.5	3.58	....	....	2.5	.....	40	....	..
8/27	8	6	11	.....	.....	....	....	.....	.....	....	....	..
8/28	8	8	10	.....	.....	....	....	.....	.....	....	....	..
8/30	8	4	+	.....	.....	....	....	.....	.....	....	....	..
9/ 1	8	4	+	0.75	0.12	....	....	.....	2.5	40	....	..
9/ 3	8	4	7	.....	.....	....	....	.....	.....	....	....	..
9/ 7	8	6	8	.....	.....	....	....	.....	.....	....	....	..
9/10	8	5	2	1	0.15	....	....	.....	2.5	40	....	..
9/13	8	8	3	1.5	0.22	....	....	.....	2.5	40	....	..
9/16	8	8	3	2	0.29	....	....	.....	2.5	40	....	..
9/21	9	..	4	2.5	0.36	....	....	.....	2.5	40	....	64
9/29	9	8	..	.....	.....	....	....	.....	.....	....	....	..
10/ 7	10	4	..	2.5	0.36	....	0.25	.....	2.5	40	....	..
10/18	11	2	4	2.5	0.35	2	....	.....	2	40	....	..
10/25	11	5	5	2.5	1.05	4	....	0.5	1.5	40	....	..
11/ 3	11	8	3	2.5	1.05	5.5	....	0.5	1	40	....	..
11/17	11	14	2	2.5	1.4	5.5	....	0.75	0.75	30	0.50	..
11/29	11	11	9	2.5	1.9	5	0.5	1.10	.....	20	0.75	..
12/17	12	6	2	.....	.....	....	....	.....	.....	....	....	..

* Plus signs indicate diarrhea, number of stools not mentioned.
† Percentage of milk and cream.

In two days, the mother thought the baby acted much better. It slept better and had less gas, but there were from six to seven stools a day, showing no change from the high protein variety.

On October 7 the baby weighed 10 pounds, 4 ounces; she was happy and slept well for the first time in her life. She had good stools, with no excess of soaps, and showed no excoriations. It was then five weeks since the institution of the low carbohydrate formula, and it was thought that the digestive tract had had a sufficient rest from lactose, the only sugar that could have caused trouble, as it was the only variety the baby had had in two months. I preferred to try a malt sugar first to test out the carbohydrate tolerance.

On October 25 it was reported that the child was very hungry, cried much and vomited a great deal. We discovered that on account of the baby's hunger the mother often gave 16 ounces at a time. We laid the vomiting to this error, and advised the mother accordingly.

The baby was doing very well, and it was considered that a little lactose now would do no harm. So I decided to add a little in skimmed milk and watch results. At the same time I increased the dextrimaltose to 4 per cent. The lactose in this mixture amounted to 1.05 per cent.

During November the buttocks were again slightly excoriated. On November 29 the baby lost 3 ounces and was constipated. It would not eat or sleep, and no underlying cause was found, although it was not thought to be an indigestion. A little malt sugar was put into the formula to help the constipation, the precipitated casein was left out, and all the protein added in the form of skimmed milk protein.

On December 17 everything was satisfactory to the mother, and she had given of her own accord orange juice, farina, bread and crackers with no ill results, although the baby was only 8½ months old.

This case seems to have been one of lactose intolerance. The baby gained and did exceedingly well when the lactose was greatly reduced beyond the means ordinarily used in treating a case of indigestion with fermentation. The dextrimaltose did no harm, even when added to 5.5 per cent. Starch did no harm either as a diluent or in the form of bread and crackers. The lactose in the final mixture amounted to 1.9 per cent. I never tried adding any extra lactose. It is interesting to note that the excoriations continued at one period while the stools were of the high protein variety and alkaline.

Having had success with this rather obstinate case, I attempted to treat other cases of indigestion with fermentation in this same manner. I shall report these very briefly.

Baby H. came to us on October 13 weighing 8 pounds at 6 months, with an illness of from ten to twelve stools a day and excoriations. The baby was immediately started on a very low lactose formula, similar to that described in the Baby D. case, and in five days the excoriations were gone and the baby was constipated. In the first nine days it gained 13 ounces. Dextrimaltose was gradually added and skimmed milk protein gradually substituted for the precipitated casein, so that in six weeks the baby was on a mixture of 2.5 per cent. fat, 6 per cent. dextrimaltose, 1.25 per cent. protein, 0.75 per cent. barley starch, and the child had gained 3 pounds, an average of 8 ounces a week.

Baby G. on October 4 gave a history of frequent, loose and frothy stools. It was put on the low lactose mixture, and in the first week gained 6 ounces and was having four normal stools a day. The excoriated buttocks had cleared in three days.

Baby T. was started on September 20 on fat-free buttermilk. The child was having from twelve to twenty-four stools a day and had excoriated buttocks. After four days the condition was not improved. The baby was then started on a low lactose formula. In three days the stools numbered three a day, and in two more days the buttocks were perfectly clear. After ten days the formula was worked up from 0.5 to 2.5 per cent. fat. I was about to begin to add dextrimaltose when, in my absence, the formula was changed to 2 per cent. olive oil, 4 per cent. dextrimaltose, 1.4 per cent. protein, 0.75 per cent. barley starch. Three days later the mother reported vomiting four times a day, and twelve or more stools a day. I put the baby back on a low lactose formula, but the mother never returned for further treatment.

Baby McC. had a similar history of loose movements and excoriations. In eleven days of treatment the stools were reduced to two a day, and the excoriations were soon gone. The baby gained 17 ounces in the eleven days. Constipation occurred while building up the dextrimaltose, and was still present after two and one-half months of treatment. The baby gained 2½ pounds in the two and one-half months. It was taken off the precipitated casein after four weeks of treatment.

Baby F. came down from ten stools to one stool a day in one week, and gained 8 ounces during the seven days.

Baby T. McC. cleared up from a similar condition in three days. He was treated for five weeks, when finally taken entirely off the precipitated casein, and gained 1¾ pounds. After the third day he did not have over three stools a day.

Baby W. had loose, green, watery stools with mucus, pus and blood, which gave negative tests for the gas bacillus. Lactose solution did not help, and the child lost 18 ounces in five days. It was then put on the low lactose formula, and in nine days the stools were normal, and the baby gained 13 ounces. In four weeks more the baby was off the precipitated casein, and had gained 2 pounds 9 ounces in the four weeks.

Baby C. had three formed stools a day while being treated in the outpatient department. The buttocks, however, were excoriated. He was put on the low lactose formula, and the excoriations healed up permanently, the formula not being used after two weeks of rest from the lactose.

Baby F. had a similar history, the buttocks clearing up immediately.

Baby C. was having from fifteen to twenty stools a day with excoriated buttocks. He became constipated and the buttocks were clear in three days, neither symptom again returning. He was kept on the low lactose formula for seven days, not receiving precipitated casein after that.

Baby W. had excoriated buttocks while being fed in the outpatient department. I gave him the low lactose formula, and at the next visit in six days the excoriations were all gone. Another attendant put him back on a moderately high lactose formula, and the baby soon became excoriated. He probably did not have a long enough rest from the carbohydrates.

Baby S. developed loose stools and excoriated buttocks while being treated in the outpatient department. She was put on the low lactose formula, and became constipated in four days. The excoriations were gone.

Baby S. cleared up from loose stools and excoriated buttocks in twelve days, having gained 4 ounces during the time.

Baby S. had a diarrhea for two days from too much Mellin's food. He was not excoriated. He was put on the low lactose formula and became constipated immediately.

I have cited here fifteen babies that did well. Eight others were treated with this formula, three not coming back for further advice. In two of the other five the treatment was interrupted in one by pneumonia, in the other by accidental poisoning with arsenic. In one

case the mother became discouraged because of the outpatient routine. She desired to have the baby treated immediately. The baby was not very sick and I learned later from the social service nurse that it was gaining slowly on milk and water.

In another case we were forced to give cane sugar because the baby absolutely refused food without it. This caused delayed recovery, but after two months the child was well and had gained 1¼ pounds. It had more excoriations than any baby I have ever seen. These cleared in two weeks. The last patient in my list did not improve, and the mother admitted she had been giving other milk because the child was hungry.

I recently looked over about 100 cases of indigestion with fermentation from the outpatient department records of the last fifteen months previous to my series. I found that almost invariably the treatment had been a formula containing from 2 to 4 per cent. lactose with no other sugar added. In some cases, buttermilk was given because a positive test for gas bacillus was found.

It was very difficult to obtain in figures the exact number of days necessary to clear the diarrhea and excoriations in each case of this earlier series. Very often the condition of the bowels or the buttocks was omitted from the record. Many times the patient was absent for two months or more after treatment, and no mention was made of the old condition in the new note.

On the whole, the cases of indigestion with fermentation, when the treatment consisted of from 2 to 4 per cent. lactose, were much longer drawn out than when the treatment was by the very low lactose formula. A point to guard against is the possible loss of weight during the first two or three days because of the low caloric content of the formula. Very often the baby will refuse the food at first, because it contains nothing sweet. The mother should be warned about these two possibilities from the start, and should be encouraged by the rapid clearing of the buttocks and diarrhea. In only three cases was the mother's confidence apparently lost.

Another difficulty in using the cream and precipitated casein mixture is its preparation. The higher the percentage of cream used, the lower will be the lactose content of the formula. As for making precipitated casein in the home, it is almost out of the question. There are on the market preparations of powdered precipitated casein which dissolve readily in hot water, and leave too slight a sediment or curd to block up the hole in the nipple. The precipitated casein made from skimmed milk and not dried and powdered is of course insoluble, and remains in curd form at all temperatures. This blocks the hole in the nipple and requires a great deal of trouble at each feeding. Even

putting the curd through the homogenizing machine does not relieve the difficulty, although it makes the curd finer.

If a 32 per cent. cream is obtainable, the powdered casein may be purchased at a low cost, and the formula prepared at home. In Boston the mixture is readily prepared on order by either the Walker-Gordon or Hood laboratories.

On the average it should take from two to four weeks to carry a child to a point at which the carbohydrate is again raised to 5 or 6 per cent., and the precipitated casein omitted from the mixture. As an increase in the protein from skimmed milk necessitates the addition of lactose, the protein should be kept low for a long time, from 1 to 1.25 per cent.

Nothing is claimed for the precipitated casein in this method of feeding except that it makes possible a very low carbohydrate food, it being almost lactose free. Whether the reduction of the soluble salts brought about by its use is of any benefit I am not able to say. I do not think the precipitated casein helps because of the preponderance of protein fermentation that it affords over that of carbohydrate, because this balance is soon reversed when the fat and dextrimaltose are raised, with no return of the symptoms. In the case of Baby D. the protein fermentation exceeded the carbohydrate fermentation, as the stools were alkaline, but the excoriations and loose movements continued.

There are many cases of indigestion with fermentation brought about by an excess of cane sugar or malt sugar. These are relieved by dispensing with the disturbing sugar. It is my opinion that the symptoms would clear up faster and surer if all the carbohydrates were reduced to a minimum for a while, and then a different one gradually substituted for the one that caused the disturbance.

In treating fat intolerance we try at first to give a food free from fat, and gradually build up a tolerance. It seems, therefore, reasonable in treating carbohydrate indigestion to get the carbohydrate as low as possible for a while, and gradually build up a tolerance.

It is interesting to note in my series how well each baby took fat up to 2 per cent., 2.5 per cent., and even 3 per cent., while on the low lactose formulas. This method may be a help in establishing a fat tolerance in some cases.

Of what use such a formula may be in the noninfectious diarrheas of the summer months I am not able to say. As most methods so far have led to some discouragement, perhaps this one may prove valuable.

272 Newbury Street.

SUGGESTION AS A THERAPEUTIC MEASURE IN NOCTURNAL ENURESIS *

FRANCIS LEE DUNHAM, M.D.
Phipps Psychiatric Clinic, Johns Hopkins University, Medical Department
BALTIMORE

Failure to control the bodily functions of excretion constitutes a breach of conduct so offensive to civilized society that its origin as a social refinement is apt to be obscured. Although it is "only necessary to scratch civilization to find savagery," there is a tendency to forget the influence of traditional associations on civilized activities.[1] Primitive impulses, the utility reactions of a simple environment, still survive. Their readjusted nervous mechanisms are generally less stable than the reflexes of instincts subject to less direct social scrutiny. All the newer requirements of civilization call on the organism to develop self-control. Consequently many customary actions of frequent repetition speedily become unconscious mechanisms. Hand in hand with this automatic activity goes an increase in the emotional value of its omission, still more of the performance of such activities contrary to custom.[2] In the mature individual lapses in control of the urinary bladder suggest at once a psychopathic taint, for even positive, somatic disorder rarely imposes loss of control beyond the power of voluntary modification. Although nocturnal enuresis during the first two years of life is considered physiologic, training in regular habits during the first year may accomplish much. If by the end of the third year restraint has not become habitual the situation should be looked on as one demanding more than mere nursery attention. Few cases of uncomplicated enuresis diurna are seen in children of ordinary intelligence, but sleep presents conditions to which certain of these ill-balanced children are unable to become adjusted. As a result various neurotic manifestations develop, sleep walking, night terrors, bed-wetting, all rightly regarded as evidence of nervous instability. The annoyance that a child disposed to bed-wetting causes itself and its attendants is so much greater than that resulting from other neurotic symptoms that its frequency is naturally overemphasized. Even among nervous children its characterization as one of the commonest diseases of childhood[3] is not substantiated by experience. Among 800 nervous children of various

* Submitted for publication June 6, 1916.

1. Boaz, F.: The Mind of Primitive Man, Journal of American Folklore, 1901.
2. Baldwin, J.: Social and Ethical Interpretations, New York, 1902.
3. Mello-Leitao: Treatment of Nocturnal Enuresis in Children, Brit. Jour. Child. Dis., 1912.

nationalities admitted to Phipps Psychiatric Dispensary, ranging in age from 5 to 16 years, only 7 per cent. were bed-wetters. Various theories point to its obscure causation, this suspicion being intensified by an analysis of the cases. Admitting the occurrence of a purely objective type, due to congenital defects, intestinal parasites, diabetes, cystitis and other organic disturbances, there is another group of subjective cases of an entirely different character. In these children evidence of psychic inferiority may be easily elicited or it may be quite elusive. Consequently they are often dismissed as habitually nervous, their malady to be outgrown. Successful treatment of these patients with empirical remedies is frequently as surprising as it is inexplicable. The element of mystery thus added temporarily enhances the virtue of the cure until its failure for each and every case relegates it to the limbo of the charlatan, another nostrum taking its place.

Without attempting thoroughly to analyze a situation presenting many physiologic elements of controversy, a brief recapitulation of the definite, biologic manifestations bearing on the cases here clinically reported is offered for criticism. The earliest response of the human organism to bladder stimulation is an involuntary process. During the first few months of life it represents the activity of a purely unconditional,[4] spinal reflex, the mechanism of which resides in the sacral and thoracic autonomic systems.[5] Awake or sleeping the normal infant exerts no attempt at voluntary control. Even after he is able to associate a manifest degree of discomfort with his unpleasant habit he is unable to readjust himself to the situation without assistance. Whether the muscular structures at the neck of the bladder are stimulated by chemical or physical agents the subsequent reflex activity seems to be touched off by the stimulus of the few drops of water in the posterior urethra.[5] The primitive nature of the mechanism is shown in the fact that section of the thoracic-autonomic innervation does not affect the reflex, though it is abolished when the pelvic nerves are cut.[6] This difference in function as well as in later dissociation phenomena might be explained on the ground that the two systems have an independent, biologic history of dissimilar chronology.[7] Thus, early reflex activity, controlled wholly by instinct, independent of cortical association, reveals the regularity of primitive organic response to environment, through the nervous system. Moreover, one may recognize herein another function of nervous tissue, that of forming new reflexes in

4. I have called these reflexes of recent formation "conditional reflexes," in order to distinguish them from the ordinary reflexes to which I have given the name "unconditional reflexes."—*Pawlow.*

5. Luciani, L.: Human Physiology, Translation, London, 1913, ii.

6. Elliott, T.: The Innervation of the Bladder and Urethra, Jour. Physiol., 1907.

7. Langley, J.: The Autonomic Nervous System, Brain, 1903.

order to adapt the organism to its social environment. These reflexes of recent formation, or conditional reflexes, together with the cortical, association neurons or analyzers, are responsible for all higher, nervous activities. They represent the reaction of the organism as a unit in response to a localized stimulus. They are phenomena of a higher, more complex order than are simple contractions or other direct changes in the motor organs at the point of stimulation. Pawlow's experiments on animals indicate that fully formed, conditional reflexes exhibit great sensitivity to all sorts of conditions, on which account they are subject, in circumstances of everyday life, to continued variation, often to complete inhibition.[8] Krasnagorski's[9] observations show the same results in children. Three distinct types of inhibition have been recognized: one conditioned by drowsiness and sleep; another due to the synchronous arrival, at the higher centers, of extrinsic and intrinsic stimuli giving rise to other reflexes; a third due to disharmonies between conditional and unconditional stimuli from which the reflex was formed. Returning now to events in the life of the unconditional or primitive spinal reflex concerned with urination, one finds that the educational procedure adopted by the attendant induces voluntary control in the child by presenting frequently a temporary association stimulus coincident with the biologic stimulus. The child attends to the nursery chair or other associative object at the same time that the desire stimulus is about to react. Through this cooperation he is enabled gradually to build up a definite control, stimulated by the visual or auditory image associated with the accomplishment of desire. These temporary stimuli or associations, presented repeatedly in conjunction with the unconditional stimulus inducing desire to empty the bladder, make for themselves new inhibitory paths. Impulses which formerly went to a particular region of the nervous system now become directed to a different one, thereby inducing controlled action in the nature of a conditional reflex. Thus the child, at first forced to conform to rules of conduct, acquires a habit thereafter voluntarily cultivated. This is all the more easily directed or diverted since "the bladder is very sensitive to reflex stimulation, every psychical act and every sensory stimulus being apt to cause contraction or increased tone in its wall."[10] The new reflex formed has a different afferent neuron, "the voluntary act of urination being essentially a reflex through the central nervous system."

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8. Pawlow, I.: The Investigation of the Higher Nervous Functions, Brit. Med. Jour., 1913.

9. Krasnagorski, N.: Ueber die Bedingungsreflexe im kindesalter, Jahrb. f. Kinderh., 1909.

10. Howell, W.: Textbook of Physiology, Philadelphia, 1915.

Elliott has observed that the pelvic nerves cause the bladder to contract. They also inhibit the sphincter, hence they are the nerves of micturition. They affect the primitive visceral movements that are the first need of the animal, to wit, the voidance of the excreta. It seems to be a reasonable assumption that they are the aboriginal nerves. The hypogastrics represent later refinements, whereby these acts may be deferred to a moment better suited to the other activities of the whole animal. They are the nerves which facilitate retention of urine and justify the belief that this second condition of the muscle has been recently developed.

This act consciously acquired and assiduously practiced is gradually relegated from a wholly conscious to a largely unconscious performance. In the infant, whose associative mechanism is undeveloped, no voluntary control is possible until there is a linking up of the vegetative and associative nervous system. After the age of 2 years the unconditional mechanism is firmly established in the normal child as a conditional reflex. The association between motor activity and its emotional response thus occurs more or less faultlessly in the absence of conscious supervision. The success of the process depends, of course, first, on an ability consciously to attend to the stimulus—sensation, desire—which underlies the motor impulse, together with an ability to interpret these sensations of behavior in terms of conduct; second, an ability unconsciously to interpret these phenomena. For "eager desire one must substitute attention,"[11] a condition which can be realized only in individuals possessing a fairly well-balanced mentation. Thus, children who show a marked disagreement in mental and chronologic age are unsatisfactory subjects. Those who respond most readily to constructive treatment are intelligent though ill-balanced individuals. Again, success in education depends on the anatomic continuity of the newly formed reflex arc, this in turn depending on the permanency of the synapses. Disjunction most readily occurs in newly formed association centers, the autonomic system being especially vulnerable from the fact that no efferent fibers of the system run from the central nervous system to the bladder without having a nerve cell in their course.[12] Thus fatigue, to which conditional reflexes are sensitive, or the vagaries of the ductless glands, with whose functioning the autonomic system is intimately associated, may readily induce synaptic disjunction. In case of this lack of associative activity the primitive condition easily dominates the situation, thus abolishing voluntary control. Elliott's observation that epinephrin, however applied to the bladder never causes any effect other than that of relaxation may thus

11. Pawlow, I.: The Work of the Digestive Glands, London, 1910.
12. Langley, J.: Nerve Fibers of the Bladder, Jour. Physiol., 1911.

explain the theory of Konrádi,[13] Williams[14] and others that the underlying cause of nocturnal enuresis is always a glandular disharmony. The favorable action of atropin on many of these cases[15] may also be accounted for in its sedative affinity for the autonomic fibers. Applying Pawlow's observations on inhibition to the reflex in question, we may make a logical explanation of the hypothesis of Fürstenheim,[16] Collin[17] and others who regard "the soundness of sleep as the initial factor," or with Herrman's[18] classification of bed-wetting with tics and speech defects, or with Pronstein[19] whose examinations reveal constant anatomic lesions.

In this group of ill-balanced children, whose unstable nervous mechanism is indicated by the tendency to bed-wetting, one is confronted with a problem of biologic adjustment. Success depends on the ability to change a situation which, during sleep, simulates a primitive state controlled by an unconditional, reflex mechanism. Granted favorable circumstances, all that is required for a positive effect is the ability to awaken attention, thereby establishing associative control through a conditional reflex. By presenting repeatedly a definite stimulus the bladder is reeducated in a proper method of functioning. The form in which this associative suggestion should be presented depends on the point of view of the observer. It certainly does not present a new or unique method of procedure, for it is narrated in the Saxon Chronicle that the Magi also taught the patient suffering from this disorder to drink the ashes of a pig's pizzle in sweet wine, and so to make water into a dog's kennel, adding the words: "lest I like a hound, should make urine in my own bed."[20] Bruni[21] reported "twenty-three cases treated by injection of normal salt solution into the arachnoid sack, with lumbar puncture, seven of which were cured." Allaria[22] reported "pseudo-epidural injections of normal salt solution under the skin, merely, with the same success as when injected into the

13. Konrádi, D.: Die Organotherapie der Enuresis nocturna, Pest. med.-chir. Presse, 1910.

14. Williams, L.: Adenoids and Nocturnal Enuresis, Brit. Jour. Child. Dis., 1909.

15. Simpson, J.: Incontinence of Urine in Children, Edinburgh Med. Jour., 1913.

16. Fürstenheim: Enuresis nocturna infantum, Therap. Monatsh., 1908.

17. Collin, A.: Contribution à l'étude de l'énurésie dite essentielle, Gaz. d. hôp., 1911.

18. Herrman, C.: The Treatment of Enuresis by Reeducation, Arch. Pediat., 1910.

19. Pronstein, R.: Russk. Vrach., 1914.

20. Cockayne, O.: Leechdoms, Wortcunning and Starcraft, London, 1864.

21. Bruni, C.: Il metodo Cathelin nella cura dell' incontinenza essenziale d'orina, Atti d. r. Accad. Med.-Chir., di Napoli, 1905.

22. Allaria, G.: La punctura pseudo-epidurale nell'enuresi essenziale dei bambini, Pediatria, Napoli, 1912.

sacral canal." Rotch,[23] following an old precedent, advocated "raising the foot of the bed so that the urine shall not irritate the neck of the bladder." Various clinicians, resorting to placebos of bitter decoctions, by instructing the child as to administration and results, report cures more or less permanent. Punishment sometimes works wonders. In spite of a degree of success, these methods, all of which are frankly modifications of suggestion, are open to criticism. One is justified in shrinking from surgical procedures if equally satisfactory results may be attained through less objective methods. Again, in spite of the harmlessness of a placebo, the possibility of drug habits, especially in children of neuropathic constitution, is to be considered in choosing a therapeutic method.

As a harmless, and, in the experience of the writer, a uniformly efficient means of treatment, the following technic in applying suggestion to these cases is presented. A set of four associative sentences is printed on a card. The wording is not immaterial, though it may be varied to suit the case. It should be positive and of simple structure. Following are the stimulus words used for the cases here reported:

I am not going to wet the bed.
I am going to wake up at midnight.
I shall get up and pass water.
I shall not wet the bed any more.

This is to be repeated ten times, twice a day, preferably during the act of urination, and at bedtime just before going to sleep. The card is then put under the child's pillow. This method, combined with no other measure, was employed in treating the ten patients whose cases are reported below. Auditory-motor stimuli were at first used, with satisfactory results in high-grade children, but were less applicable to children of moderate responsibility. The visual-motor method has been adopted, however, as a better routine procedure. The subjects were all schoolchildren who, from their appearance and conduct, were tacitly neurotic. Treatment was begun Feb. 1, 1916, and is being continued. The results here reported are therefore of four months' duration. The two girl subjects were at home while all the boy subjects were inmates of the Baltimore Parental School, an ideal institution of its nature. Each subject had a thorough physical and mental examination in order to rule out etiologic factors other than those of psychic inferiority. The urine was examined in each case and was within the range of normality respecting quality and quantity. The majority had been under unsuccessful medical treatment.

Various historical factors are shown in the table of clinical data and the results are there briefly indicated. In general, one may say

23. Rotch, T.: Pediatrics, Ed. 5, Philadelphia, 1906.

CLINICAL DATA OF TEN PATIENTS TREATED FOR NOCTURNAL ENURESIS

Name	Sex	Chronological Age	Binet-Simon Age	Nocturnal Enuresis	Remarks	Results
H. N.	M	12	9	From birth, nearly every night	An ill-balanced truant; neurotic family history; cooperative, eager to stop "floating"	Perfect for 2 months; left school and lost sight of; wakes up himself
E. T.	M	12	12	From birth, nearly every night	An ill-nourished, intelligent, cooperative truant; poor home environment; psychopathic family history	Twice in February, not since; wakes up himself
O. H.	M	14	14	From birth, 2 or 3 times weekly, till 8 years, when truancy began; every night since	Highly emotional, somnambulist, snake dreams and terrors; family history of drink and debauchery	Three times only; wakes up himself
R. S.	M	11	11	From admission to Parental School 6 months before; every night; before occasionally	Bright boy, no other neurotic traits; family history good; truancy, from bad neighborhood	Twice only; wakes up himself
L. K.	M	14	8	From infancy every night	Dull boy; family history not known	Improved, but still about once a week; has to be awakened sometimes
C. P.	M	12	8	From infancy every night	Fairly bright boy; family history not known	Once in each month for 2 months; paroled and lost sight of
R. W.	M	12	12	Twice weekly from birth; every night since admission to Parental School, 6 months before	Bright boy; family history good	Only once since beginning; wakes up himself
W. W.	M	13	13	Occasionally all his life	Bright boy; poor environment	Once only from beginning
B. E.	F	9	9	Father drinks, somnambulist, bed-wetter; mother "fits and spells"; home environment "the worst"	Nocturnal and diurnal enuresis began after diphtheria at 5 years; every night; night terrors; a brother has same habits	Much improved; one month perfect; later "forgot" and wet bed once or twice a week
H. B.	F	12	12	Family history good; mother rheumatic	Nocturnal enuresis since scarlet fever at 5 years; nearly every night; night terrors; large, overdeveloped; infantile; type, normal sella turcica; choreiform movements	On second night of trial wet bed; not once since; wakes up herself.

The eight boys were observed at the Baltimore Parental School. They were all white, and were all committed for truancy.

that the mass result was entirely positive. This is evident when it is recalled that all these subjects were chronic bed-wetters, the act occurring, in the majority of cases, every night. Under treatment cessation of the habit was essentially complete in all but one case, L. K., whose mental level explains his irresponsibility and poor success. As Dubois long ago suggested, "enuresis may be cured by education only, interrupting the child's sleep for this purpose in the middle of the night. It is thus that good habits are created." It is of interest, however, to note the result of this dictum on one of these subjects, H. B. For several weeks the girl was nightly aroused from sleep by her mother and urged to micturate; rarely would she awaken so thoroughly as to recall the incident the following day. The result was negative, bed-wetting generally occurring as before, but later in the night. This well illustrates the part played by consciousness in a successful reaction. The child must assume the responsibility of waking up and caring for himself.

SUMMARY

From this point of view uncomplicated nocturnal enuresis is an element in the psychic regression of an unstable organism toward a more primitive biologic state.

The mechanism concerned in inhibition is an unconditional reflex which through social evolution becomes a conditional reflex.

The object of treatment is to assist normal development in the associative mechanism, tending thus to induce a conditional reflex.

The means employed are the presentation of a series of associative visual stimuli.

Ten clinical cases are presented.

PROGRESS IN PEDIATRICS

REVIEW OF THE LITERATURE OF RESPIRATORY DISEASES OF THE PAST YEAR

CHARLES HENDEE SMITH, M.D.
NEW YORK

RESPIRATORY DISEASES, MORTALITY AND MORBIDITY

Woodward[1] has made a study of the respiratory diseases as a factor in the causes of infant mortality. In 1913 in the first year of life the diseases of the respiratory system ranked third in the causes of death; 15.85 per cent. of the total deaths were due to respiratory diseases; 25 per cent. of the mortality from respiratory diseases at all ages occurred in the first year of life. Bronchitis and pneumonia caused 96 per cent. of the deaths by respiratory diseases in infants. The mortality in cities was slightly higher than in the country. The third week of life was the maximum danger period for bronchitis and pneumonia. He concludes that prevention of death in infancy from respiratory diseases is a task which requires the united efforts of the prenatal nurse, obstetrician, midwife, pediatrician and infant welfare station.

Haynes[2] believes that vital statistics do not measure the importance of respiratory diseases, since most patients recover, but a vast amount of damage is done which is not shown by the death rate. The ordinary infectious cold results in ear, kidney and cardiac damage of no small amount. From November to June the bulk of pediatric practice is due to respiratory diseases. Forty per cent. of the deaths in the first five years are due to respiratory conditions and 25 per cent. of all deaths from respiratory diseases are in the first five years. Rickets, infectious diseases, enlarged adenoids and tonsils all predispose greatly to respiratory infection. There are certain anatomic factors in the young which render any respiratory inflammation serious; that is, the narrowness of the respiratory passages, nose, larynx, bronchi, which are easily obstructed by the secretions, the shape of the thorax, the soft ribs, and the habitually recumbent position. Lowering of resistance by cold, fatigue, autointoxication or poisoning by food or chemical substances are also important predisposing factors. The actual infection usually takes place by spray from the mouth and nose discharges of an infected person. The helpless infant must be protected from dust, both outdoors

1. Woodward, W. C.: Am. Jour. Obstet., 1916, lxxiii, 362.
2. Haynes, R. S.: Arch Pediat., 1916, xxxiii, 81.

and indoors, from smoke which is harmful as a mechanical irritant, and from crowding, since close contact increases the possible transmission of infection by sneezing and coughing.

In the regulation of infectious diseases, early quarantine is more important than disinfection and fumigation afterward. Crowding is especially dangerous in hospitals, and the box system, or screens, is a great aid in preventing transmission of infection. The early exclusion from school of every child with a slight cold is absolutely necessary to prevent transmission of colds, not only to his schoolmates, but to the smaller children in their various families. The discharges from the nose and mouth of a sick child should be carefully caught on gauze which should be burned, and the bedding, etc., disinfected.

Among preventive measures are careful feeding, enough sleep, proper clothing, a clean skin and careful regulation of the bowels. The air in the house should be kept at 65 F., with a humidity of 60 per cent. Young children should not be taken out on windy and damp days.

Hill[3] states that epidemics of colds are most common when the humidity is great, the temperature variable but cold, in raw weather with thawing snow or cold rain and wet ground. Persons living in the open air are free from colds. Children during their holiday paddle all day in the sea with bare legs. When they are shut in the schoolroom, colds begin. This is probably due to propinquity, since children are nearer together indoors. Out of doors the sprays of saliva and nasal mucus are blown away and are less likely to come in contact with healthy children. The colds may be caused by bacterial infections, dust, irritating fumes, pollens, etc. Hill has made observations on the nasal mucous membrane and has found that out of doors it is pale, taut, and does not pit on pressure with a probe. Indoors it is swollen, congested, covered with thick secretion and pits on pressure. He thinks this is due to the fact that outdoors the air nearer the ground is warmer because it moves less rapidly than at the level of the head. Indoors the reverse is true. The floors are cold and drafty and the head is immersed in hot, stagnant air. He believes that the nasal obstruction is due to chilling of the feet when the head is warm, and has found that warming the feet will relieve the congestion observed indoors.

Ideal conditions would give a warm floor, radiant heat and abundant cool air in motion. Open fires approach this more nearly than any other method of heating, since the feet can be warmed by radiant heat and most of the heat goes up the chimney. He believes that the boggy condition of the nasal mucous membrane predisposes to infection by bacteria. Sudden changes from the warm indoor air to the colder air

3. Hill, L.: Brit. Med. Jour., 1916, i, 541.

out of doors are bad on account of the sudden demand for more blood in the nose which is necessary to compensate for the cooling of the mucous membrane and the more rapid evaporation from its surface.

Peiper[4] believes that respiratory infections in infants come not from chilling alone, but from infection from other persons. Infections are not always influenzal, though "conveniently classed as grippe." Infants should be carefully protected from these infections, as far as possible, by a room of even temperature, avoidance of chilling, and of cold drafts, especially at the bath. In raw weather a child should remain indoors. The mother should avoid infected persons and if she contracts cold or cough, must use every effort possible to protect the child from infection.

Peiper describes accurately and briefly the various infections of the upper respiratory tract, the bronchi and lungs. He also describes the treatment of the various conditions arising more fully and carefully than is usual with most foreign writers. The treatment of capillary bronchitis and bronchopneumonia are especially good. The technic of, and indications for, cold packs, mustard packs, hot baths, etc., are given in detail in excellent manner.

Miller and Noble[5] investigated the effect of chilling on the incidence of respiratory diseases in rabbits. They used rabbits infected with the *Bacillus bovisepticus,* because it is an organism normally pathologic for rabbits. They conclude that respiratory infections of rabbits are favored by chilling the animals after they have been accustomed to heat, and that the weight of evidence does not justify the elimination of cold as a possible though secondary factor in the incidence of respiratory diseases.

Marked changes of temperature from low to high predispose even more to respiratory infection than from high to low.

ROENTGEN RAY IN DIAGNOSIS

Freeman[6] calls attention to the value of the Roentgen ray in intrathoracic lesions in childhood. It is a useful aid to auscultation and percussion in clearing up the diagnosis of doubtful conditions. The Roentgen ray has its greatest value in tuberculosis, and in pneumonia without physical signs. In cardiac lesions it gives information as to the size and shape of the heart and the presence of exudate in the pericardium. In the case of a child with an elevated temperature with sibilant, sonorous, or crepitant râles through the lungs, and positive

4. Peiper, E.: Deutsch. med. Wchnschr., 1916, pp. 213 and 245.
5. Miller, J. A., and Noble, W. C.: Jour. Exper. Med., 1916, xxiv, 223.
6. Freeman, R. G.: Arch. Pediat., 1915, xxxii, 891.

von Pirquet, it is impossible to distinguish a bronchitis with small latent focus of tuberculosis from a case of miliary tuberculosis. The Roentgen ray is the only means at our command for making this diagnosis.

It is extremely valuable, also, for distinguishing effusion from consolidation, pneumothorax, etc. The thymus, bronchial and mediastinal lymph nodes often cast shadows, making a diagnosis possible. Freeman reports a case of pneumonia without physical signs, one with pericardial effusion, and a case of endocarditis in which the size of the heart showed the effect of rest and treatment.

ASTHMA

Cameron[7] believes that asthma is a respiratory neurosis. In children it is characterized more by the bronchitic than by spasmodic symptoms, and it is often impossible to determine whether a given attack is asthmatic or not by the physical signs. The following points suggest asthma:

1. Family history of asthma, gout or migraine.
2. Exudative diathesis (eczema, seborrhea, urticaria).
3. Eosinophilia.

Cameron believes that psychical treatment is very important, and that every effort should be made to avoid frightening the child during the paroxysm by overanxiety on the part of the parents. During an acute attack the room should be brightly lighted and the child should be diverted if possible by books or toys. A hot bath or an emetic sometimes relieves the attack. Chloral or atropin are considered the best drugs.

He advises a diet largely of fruits, vegetables and cereals, avoiding eggs and milk, and giving meat in small amounts only once a day. Adenoids should be removed if present. Fresh air and vigorous exercise are important. Potassium iodid should be administered. In infants treatment must be directed toward the bronchitis by confinement to the room, steam, counterirritation, etc.

Jackson[8] describes the manner in which drugs act in relieving asthmatic attacks. He divides their mode of action into two groups.

The first group acts by paralyzing the endings of the bronchoconstrictor nerves to the muscle fibers of the bronchioles, causing a relaxation of the bronchioles. To this group belong atropin, hyoscin, etc.

The second group acts by stimulating the endings of the bronchodilator nerves. This is the way in which epinephrin and allied drugs act.

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7. Cameron: Guy's Hosp. Gaz., 1915, xxix, 319.
8. Jackson, D. E.: Jour. Lab. and Clin. Med., 1915, i, 126.

It is probable that two forms of the disease may exist. These can be demonstrated experimentally. From clinical evidence it may be that these two forms may exist separately or at the same time.

The first form is due to nervous influence, central, peripheral or reflex. Nervous impulses arising from direct central influence or reflexly excited (as by a nasal sinus disease) pass down the bronchoconstrictor nerves and stimulate the muscles to contract. Possibly chemical substances in the blood may stimulate these endings directly or by acting on the ganglia in their course. We know of no such substances produced in the body, but experimentally arecoline will cause bronchoconstriction which is relieved or prevented by atropin.

The second form is due to direct stimulation of the muscle fibers of the bronchus. It may be produced experimentally by enormous doses of atropin or hyoscin. It is very evident that atropin will do no good in this type of the disease. Epinephrin relieves it by stimulating the bronchodilator endings which the atropin has not affected.

A considerable number of drugs will cause direct muscular spasm independent of innervation; for example, morphin, codein, heroin, narcotin, some of the metallic salts. A spasm caused by these drugs is more lasting than that due to the nervous form of asthma and more difficult to treat by the drugs acting on the bronchodilators.

Guinea-pigs with anaphylactic shock die chiefly from constriction of the bronchi. Atropin is of slight value in these cases, much less so than a full dose of epinephrin. It has been suggested that the substance formed in anaphylaxis is histamin or some related substance. Histamin is formed from histidin by certain bacteria. In animals histamin causes bronchoconstriction of a severe type which yields but slightly to atropin. It is possible that histamin or some related substance may be the cause of asthma in man.

Pilocarpin causes great bronchoconstriction by stimulation of the peripheral nerve endings. It is difficult to see how this drug can relieve asthma, although it has been said to do so in some cases. Possibly it does so by stimulation of the adrenal to increased secretion, and this may explain a paradoxical bronchodilatation.

BACTERIOLOGY OF NASAL INFECTIONS

Foster[9] points out that a great variety of organisms have been thought to be the cause of a common cold in the head. He has repeated Kruse's experiment with Berkefeld filtrates from the discharges of coryza. The discharges were mixed with salt solution and passed through a Berkefeld filter. The filtrate was sterile on ordinary culture

9. Foster: Jour. Am. Med. Assn., 1916, lxvi, 1180.

mediums, both aerobic and anaerobic. Ten soldiers were inoculated by dropping this clear filtrate into their noses, and nine developed typical colds in the head. Foster applied the culture methods developed by Noguchi and others, using rabbit kidney and ascitic fluid covered with oil. With this medium and the above filtrate he obtained a cloudiness around the pieces of tissue which was not obtained in controls and the repeated subcultures gave the same findings. The cultures under the dark field microscope showed small globular bodies which were apparently motile, and these were also seen in stained smears. The filtrates from these subcultures were also clear, and sterile with ordinary culture methods. When inoculated into the noses of eleven patients they produced acute coryza, and cultures from the discharges of these cases on ascitic tissue mediums gave the same clouding of the fluid around the pieces of tissue which were observed in the original cultures.

Baxter[10] made a study of sixty cases of postnasal infection in childhood, which was very prevalent in the earlier part of 1915. It was nearly all due to staphylococcus. The middle ear was involved in 75 per cent. of the cases, and this was especially common under 2 years of age. There were few other complications except cervical adenitis.

Baxter advises nasal douches with gravity pressure and the use of sodium bicarbonate or citrate, internally. He thinks that one should not be too hasty in puncturing the membrana tympani, since many cases will subside without puncture under irrigations with bicarbonate or boric acid solutions.

INFLUENZA

Leo-Wolff[11] distinguishes between influenza which is an acute infectious epidemic disease, due to *Bacillus influenzae*, and grip or grippy colds, due to pneumococcus, streptococcus, *Micrococcus catarrhalis* or Friedländer's bacillus. He believes that climatic changes, exposure to cold, or anything that lowers the vitality of the patient, predisposes to both. The mode of transmission is usually through sneezing, coughing, etc. In hospitals, epidemics may be averted or minimized by the exclusion of visitors, the wearing of gauze masks by doctors or nurses having colds, and by the separation of infants by gauze screens.

He calls attention at the fact that influenza often simulates measles, scarlet fever, meningitis and digestive disturbances in infants, and that sporadic cases are very difficult of diagnosis in such atypical cases. A general discussion of the two groups above mentioned brings out nothing of great value.

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10. Baxter, G. E.: Arch. Pediat., 1916, xxxiii, 729.
11. Leo-Wolff: Med. Rec., New York, 1916, lxxxix, 226.

PNEUMONIA

In a preliminary report of 1,000 cases of pneumonia Pisek and Pease[12] have found a mortality of 34.3 per cent.

In 445 cases of bronchopneumonia the mortality was 41 per cent.

In 227 of lobar pneumonia the mortality was 28.1 per cent.

Of the bronchopneumonia patients, the greater proportion were under 2 years of age. The mortality was 52 per cent. in the first year, and there were no deaths over 3 years.

Of the lobar pneumonia, while there was a larger percentage of the cases above 2 years of age, there was still a considerable number under that age (seventy-five in the first year and ninety-seven in the second year).

There was noted a marked difference in the variation of mortality in the epidemics of different years. The bacteria found in the lobar pneumonia cases were pneumococci with a few staphylococci and streptococci. In bronchopneumonia, streptococci, staphylococci and influenza bacilli predominated. Pneumococci with streptococci and staphylococci were also found, that is, mixed infections, and in these cases the pneumococci were usually of a low grade of virulence.

The character of the infection depended on the amount of natural or acquired resistance of the patient, the state of his vitality, local changes in the respiratory tract and the virulence of the organism.

Forty-eight cases were studied according to the type of the pneumococcus with the following results:

	Lobar	Broncho	Deaths	Death Rate
Type I	9	2	1	22.9
Type II	11	3	5	29.3
Type III	1	3	1	8.3
Type IV	7	12	4	39.8

Wollstein and Benson[13] have studied the organisms present in pneumonia in children in fifty cases to determine the type of pneumococcus present. They found Type IV in 60 per cent. of the cases, usually associated with one of the other types. The mortality was 40 per cent.

In Type I the mortality was 83 per cent., and in Type II, 33 per cent. This differs from the findings in adults in whom Type II has a higher mortality than Type I, and in whom Type IV is not often fatal.

Dunn and Hammond[14] report a case of pneumonia in a child of 14½ months, due to Friedländer's bacillus. The left lower lobe was involved, then the left upper lobe. Thoracentesis was performed on account of persistent temperature and signs at the base, and a small

12. Pisek, G. R., and Pease, M. C.: Am. Jour. Med. Sc., 1916, cii, 14.
13. Wollstein, M., and Benson, A. W.: Am. Jour. Dis. Child., 1916, xii, 254.
14. Dunn, C. H., and Hammond, J.: Interstate Med. Jour., 1915, xxii, 1133.

amount of fibrinous material was withdrawn from the lung. This gave a culture of Friedländer's bacillus, and this finding was confirmed by a second puncture. The fever persisted for eighteen days and the temperature dropped by crisis. The child made a good recovery. The case was reported in detail on account of the rarity of recovery from an infection with this organism.

Perrot[15] considers facial herpes in pneumonia or pneumococcal infections to be always a localization in the skin or mucous membrane of the pneumococcus, which is always found in the contents of the vesicle.

Herpes is often the principal manifestation of pneumococcal infection, the signs of pneumonia being ill-marked or absent. The same is true of pneumococcal septicemia, stomatitis, angina or meningitis. Infection of the skin probably takes place through the blood stream. He gives several illustrations of cases in which the herpes is the main feature of the infection.

Mason[16] confirms the findings of Weill and Mouriquand that central pneumonia does not exist; that the shadow cast by a pneumococcic consolidation is triangular in shape, the base in the axilla, the apex toward the hilum. When the apex of the bronchial shadow does not extend to the root of the lung, bronchial voice and breathing are not heard. As soon as the consolidation extends far enough to come in contact with the large bronchi at the hilum, bronchial voice and breathing are heard.

This gives a most reasonable explanation for the absence of physical signs in the early stages of consolidation and can be easily verified in any case in which daily roentgenograms are taken.

Hutinel[17] considers that pulmonary complications are not rare in scarlet fever, which is contrary to the usual opinion. In five years he observed 2,500 cases of scarlet fever, and one-third of the deaths were from pneumonia. These occurred in three forms: bronchopneumonia, lobar pneumonia and congestive pneumonia or pseudolobar bronchopneumonia.

Bronchopneumonia is frequent in small infants in severe cases with purulent rhinitis, otitis, severe angina, etc. It is streptococcal in origin and is an extension from the infection of the upper air passages. Deformities of the chest from rickets and Pott's disease predispose to pneumonia. The most severe cases are those associated with measles following scarlet, or when the pneumonia appears in an epidemic form among a group of children in a scarlet ward. These facts are well

15. Perrot, J.: Thèse de Lyon, 1913-1914, No. 130; abstr. in Brit. Jour. Dis. Child., 1916, xiii.
16. Mason, H. H.: Am. Jour. Dis. Child., 1916, xi, 188.
17. Hutinel, V.: Arch. de méd. d. enfants, 1916, xix, 57.

known, but the frequency of pneumonia is commonly underestimated, being overlooked in children who are gravely ill. If its occurrence is not borne in mind it will only be found on the necropsy table.

Lobar pneumonia occurs in three groups according to the time of its onset. Hutinel has called certain of these cases "pneumoscarlatina," which he considers comparable to cases of pneumotyphoid.

He differentiates three groups as follows:

1. The pneumonia precedes scarlet by several days.
2. The pneumonia comes on during the course of scarlet.
3. The pneumonia comes on during convalescence.

In the first form the ordinary symptoms of the pneumonia exist, and the appearance of the scarlet rash comes as a surprise. It is difficult to determine whether the rash is toxic in origin or true scarlet. The pneumonia is probably not due to the scarlet, but is an ordinary pneumococcus infection. The appearance of the scarlet does not change the diagnosis; it is merely a superadded infection. It can be determined that it is a true scarlet from the course and symptoms. The red throat, tongue, cervical adenitis, desquamation and late complications are those of scarlet. The two infections modify each other, however, and resolution is usually slower than in an ordinary pneumonia. Pleural reactions are nearly constant.

The pleurisy may be serofibrinous or a simple parapneumonic, noninfectious effusion, but usually becomes purulent. It may be due to streptococcus or pneumococcus. Some cases show pneumococcus in the sputum and streptococcus in the effusion. The prognosis is doubtful. Some patients recover, but with slow convalescence, often protracted by the late complications of scarlet.

Relapses of the scarlet fever seem to be abnormally frequent, following these cases, and may come on as late as sixty to seventy days after the original infection.

The second group, in which the pneumonia appears during the acute stage of the scarlet, is divided into two subgroups:

(*a*) In the first subgroup the pneumonia appears at the same time as the rash. It is contemporary with the angina, usually apical, and is sometimes mild and sometimes fatal. It resembles in its characters ordinary lobar pneumonia.

(*b*) In the second subgroup the pneumonia appears after the eruption has existed several days. It is usually at the bases, bilateral, and resembles bronchopneumonia. It is commonly a streptococcal infection and the prognosis is usually grave.

The pleural reactions are frequent, and vary from a transient clear effusion to an empyema of a severe type. The scarlet symptoms rarely predominate, but are generally of subsidiary importance.

The third group comprises pneumonias occurring during convalescence. These are accidental pneumococcus infections. They are not very rare and the prognosis is usually good. Pleural complications are frequent, however, and they seem to predispose to relapses of the scarlet in the same manner as the first group. In the cases complicated by empyema, prognosis is less good, 30 per cent. being fatal.

Hutinel concludes that in pneumonia and scarlet occurring together, the infections are apt to aggravate each other; the pneumonia is influenced more than the scarlet, and its prognosis is much less favorable than in an uncomplicated pneumonia. The pleural complications are frequent, varying from a mild serous effusion to severe empyema. Relapses occur in one third of the cases complicated by pneumonia. This is very much more common than in uncomplicated scarlet, in which relapses occur in not more than 1 per cent. Hutinel suggests the possibility that pneumonia modifies the effort of the organism acquiring immunity, and so makes reinfection with scarlet more common. This paper is illustrated by many cases reported in detail.

Toupance[18] considers that the respiratory complications of scarlet fever are less rare than the textbooks usually state.

He has found laryngitis in 5 per cent. of the cases, due to streptococcus. In young children it is usually fatal.

Bronchitis occurs in 2 to 6 per cent. It is commonly mild and always suggests the possibility of measles.

Bronchopneumonia is found in 1 to 2½ per cent. of hospital cases, usually accompanying severe infections of the nasopharynx or general sepsis. The mortality is 50 per cent.

Lobar pneumonia was found in 1 per cent. of the cases. It may coincide with the onset of the scarlatina and the two diseases run their course without effect on each other. Relapses are common in such cases. When it occurs during the eruptive or desquamative stages it tends to affect the apex of the lung and often runs a torpid form which is difficult of recognition. It is frequently accompanied by pleural effusion.

Pleurisy is found in 0.8 per cent. of the scarlatina cases; two thirds of these are purulent.

Of the empyema observed, some cases have been generalized, some have been "latent" (bronchopneumonia with interlobar effusion); all have been characterized by rapid pus formation, abundant effusion, and grave general condition. The death rate was 40 per cent.

Serofibrinous pleurisy has usually been of streptococcus origin. It may be associated with foci of pulmonary congestion or complicated by pericarditis or tuberculosis.

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18. Toupance, M.: Thèses de Paris, 1914-1915, No. 80.

Allan[19] reports a case of a child with abdominal symptoms indicating acute appendicitis. Operating on the sixth day of the disease, the appendix was found to be inflamed, contained a blood clot and several thread worms. The anesthetic was chloroform-ether. A cough developed the day after the operation and signs of consolidation were discovered at both bases. Two days after the operation, the eighth day of the disease, the temperature, which had been persistently high, fell by crisis. The case is remarkable on account of the definite inflammation of the appendix in a case of pneumonia, showing it was not an ordinary case of pneumonia with abdominal signs.

Caillè[20] reports three cases of foreign-body pneumonia. The first was a child of 2 years and 10 months who had been sick for four weeks with fever and chills and vomiting followed by dyspnea. There were purulent sputum and leukocytosis, with dulness at the base of the right posterior chest. The Roentgen ray showed a tack in the bronchus, which was removed by a bronchoscope guided by the fluoroscope.

The second was a child of 2 years who had had pneumonia the year before, following which there was persistent cough and physical signs (flatness over the chest, bronchial breathing and râles). The Roentgen ray showed a nail in the left bronchus and trachea. An effort was made to remove this with the bronchoscope, but this was unsuccessful. A second attempt through a tracheotomy wound was successful, but pneumonia resulted and the child died.

The third was a 4-year-old child who swallowed a shawl pin three days before admission. The Roengen ray showed the pin to be in the left bronchus. An attempt was made to remove the pin, but this was unsuccessful and following this there was a fever which lasted two weeks. The pin was finally coughed up forty-three days after it was swallowed.

Caillè emphasizes the importance of using the Roentgen ray in unresolved and atypical pneumonia. He urges that all foreign-body cases be reported in the lay press as a warning to parents to keep objects which might be inhaled or swallowed out of the reach of small children. The indirect suggestion offered children by seeing parents put things in their own mouths may lead to imitation by the children. It should, therefore, be avoided.

Clarke[21] reports his experience in about thirty cases of pneumonia treated with hexamethylenetetramin. In every case except one the temperature started to fall a few hours after beginning the drug, and in

19. Allan, J.: Brit. Jour. Child. Dis., 1916, xiii, 207.
20. Caillè, A.: Arch. Pediat., 1915, xxxii, 881.
21. Clarke, T. W.: New York State Jour. Med., 1915, xv, 445.

every case the patient became well in twenty-four to forty-eight hours. The drug was given in most cases during the first four days of the disease. There was one bronchopneumonia resulting in death in an infant of 4 months.

Whenever a child has failed to respond in forty-eight hours he has always been able to find a complication. He also has given the same remedy in influenza, measles and pertussis and has had no pulmonary complications. He pushes the drug to the limit of tolerance, well diluted in water or milk. Occasionally strangury or hematuria results, in which case the drug is discontinued and sodium bicarbonate given.

In a clinical lecture on the treatment of bronchopneumonia, Marfan[22] advises that the sick-room should be kept at 62 F., with free ventilation, but avoiding chilling the patient. The child's position should be changed frequently to avoid congestion at the bases.

Nasal catarrh should be treated by instillations of eucalyptol in oil, 1 to 6, or collargol, 1 per cent.

If there is no digestive disturbance, the child should be fed "as in any other febrile state with equal parts of milk and sweetened water."

He differentiates four forms of the disease, acute, hyperacute (capillary bronchitis), subacute, and latent. He advises counterirritation by hot baths at 100 F. every three hours when the temperature reaches 102.5. These provoke vasodilatation, relieve the dyspnea and promote sleep. Wet packs at 80 F. can be given from the arm pits to the umbilicus for one and a half hours. The nondepressing expectorants such as ergotin and nux vomica may be used.

In influenzal cases he recommends inunctions of collargol, 3, lanolin and petrolatum of each, 10 parts.

In the suffocation attacks of the hyperacute form, mustard baths or mustard packs should be used and stimulation with camphor, caffein and oxygen are necessary.

COLD AIR IN TREATMENT

Morse[23] differentiates between the value of fresh air and cold air in the treatment of respiratory infections in childhood. Discomfort in a closed room is not due to expired poisons, but to warmth, moisture and stagnation of the air. Fresh air is air that is cool, dry and in motion. Dust is undesirable.

It is possible to lay down the following rules from clinical experience:

In nasopharyngitis cold air is irritating in the early stages and predisposes to otitis. Fresh air is of advantage, but it should be warm.

22. Marfan, A.: Med. Press and Circular, 1915, c, 408.
23. Morse, J. L.: Jour. Am. Med. Assn., 1916, lxxiii, 375.

The child should be kept in a room at a temperature not below 60 F., if he is kept in bed, or 65 to 66 if out of bed.

In laryngitis, cold air is very irritating in the early stages, but has no effect later. Patients are more comfortable if the air is moist, and at about 70 F.

In acute bronchitis the cough and sternal oppression are increased by cold air in the beginning of the disease. Children should be kept at a temperature of 60 to 70 F., and the air should be moist. In later stages cold air is less irritating, but has no advantage over warm air. It is no longer necessary to have the air moistened, as the mucous membrane of the bronchi is covered with secretion.

In bronchopneumonia the acute stage of the bronchitis is usually passed, and hence there is no contraindication to the use of cold air.

In lobar pneumonia there is usually no bronchitis and here cold air has its greatest value. There is no question that the general stimulating effect of the cold is of great advantage. The patients are much more comfortable, but the available statistics show but little difference in the mortality in children treated in cold air.

Freeman[24] believes that the pediatrist has a powerful therapeutic agent in moving, cool outdoor air. It stimulates the appetite, induces quiet sleep, brings color to the cheek, increases the resistance to infection.

Howland and Hoobler's observation that the blood pressure is raised by a change from a warm room to cold air has not been confirmed by later observers, and so the good results cannot be attributed to the effect on the circulation. The favorable action of fresh air is probably due to the absence of the depressing effect of the air in closed rooms. It has been shown that this is not due to diminished oxygen or increased carbon dioxid in the air, but to the fact that stagnant, warm, moist air prevents cooling of the body. In cool fresh air the body can cool itself more easily. Outdoor porches are best in the treatment of sick children and are probably applicable to all infectious diseases except possibly scarlet and measles. Fresh air finds its greater use in pneumonia and tuberculosis. The best substitute for the sleeping porch is a room with cheese cloth screens in the windows.

Premature infants should be kept in air which is warm enough to keep up the body temperature, but it should be freely moving warm air, not stagnant.

In renal and cardiac cases the value of cold air is doubtful. Freeman believes that parents and children should be thoroughly trained to the value of fresh air and away from the idea of the harmfulness of drafts.

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24. Freeman, R. G.: Arch. Pediat., 1916, xxxiii, 330.

Cunningham[25] has analyzed 1,500 cases of pneumonia treated in the Children's Hospital in Boston. Two hundred of these patients were treated prior to 1906 in a fairly well ventilated ward. After 1906 all patients were treated out of doors. Cunningham concludes that the results of open air treatment have not come up to expectation and that the death rate is slightly higher than before the cold air treatment was instituted. The patients were formerly treated in a ward which was well ventilated with a good grade of fresh air, although it was warm. Empyema has been slightly more frequent under the cold air treatment. Before 1906 the duration was 9.1 days. Since then under the cold air treatment it has been 8.7 days.

These statistics are interesting in view of the almost general use of cold air in treatment of pneumonia.

Freeman,[26] on the other hand, states that it is an accepted fact that fresh air increases the appetite and digestive power, and in spite of its doubtful effect on the blood pressure he believes that fresh air is of the greatest value. In his twenty-five cases of lobar pneumonia there were only 12 per cent. of deaths, and in the uncomplicated cases only 4.7 per cent. In sixty-two cases of bronchopneumonia there were 16 per cent. deaths and in the uncomplicated only 3.3 per cent.

Morse and Hassman[27] have made a large number of blood pressure readings on children with pneumonia in an attempt to verify the work of Howland and Hoobler. They found that the temperature of the surrounding air had no effect on the systolic, diastolic and pulse pressures. The temperature of the surrounding air seemed to affect the pulse rate, however, the pulse being lower out of doors than in the ward. The same is true of the rate of respiration, although there were many exceptions to both of the latter observations.

The general impression of the physicians and nurses was that the children were more comfortable outdoors than in the ward. They coughed less, were quieter, had a better color and took their food better out of doors.

No conclusions are justified at present as to the effect of the cold air treatment on the mortality in pneumonia.

PLEURA

Gittings, Fetterolf and Mitchell[28] have found that the fissures of the lungs show practically the same relation to the bony framework of

25. Cunningham, A. R.: Boston Med. and Surg. Jour., 1916, clxxiv, 753.
26. Freeman, R. G.: Am. Jour. Med. Sc., 1916, cii, 1.
27. Morse, J. L., and Hassman, D. M.: Am. Jour. Dis. Child., 1916, xii, 445.
28. Gittings, J. C., Fetterolf, G., and Mitchell, A. G.: Am. Jour. Dis. Child., this issue, p. 579.

the chest in infants as in adults. The variations are considerable, but do not depend on any anatomic character of the chest, and therefore cannot be predicted from the shape of the chest. The lower level of the lungs is not quite so low as in adults, and hence great care must be taken to avoid injury to the diaphragm in thoracentesis. The lowest safe point is in the fifth or possibly the sixth space in the midaxillary line, and the seventh or possibly the eighth space in the line of the angle of the scapula. The optimum point of puncture is the seventh space in the posterior axillary line.

Bayne-Jones[29] reports a case of pleural eosinophilia following pneumonia. The case presented no unusual features and was a pneumococcus infection of Type II. It was followed by pleural effusion. The fluid was sterile. It contained 580 to 600 cells, of which 6 per cent. were polymorphonuclear and 45 per cent. eosinophils. The blood contained only 1.2 per cent. eosinopils. Pleural eosinophilia is a comparatively rare condition, but is probably more common than is usually thought to be the case on account of the failure to stain the smears with polychrome stain. Bayne-Jones has found an incidence of from 1 to 5 per cent. of a series of patients who were examined carefully in regard to this point. It has been reported in eight cases following a lobar pneumonia. It has been found in a long line of diseases, including practically every cause of pleural effusion. There is no relation to the cytologic formula of the other cells in the fluid, or to its sterility. It has no diagnostic significance except that the effusion is usually nontuberculous. The prognosis is good, since these effusions are nearly always transient and benign. The only explanation offered is that it is possibly a local anaphylactic reaction, from the splitting of the proteins occurring during the process of resolution and absorption.

EMPYEMA

Brown[30] says that the recognition of pleural disorders by the Roentgen ray is rendered difficult by the fact that in acute pleurisy, especially if complicated by pneumonia, the patient cannot be brought to the Roentgen-ray plant. A portable outfit is necessary and the portable fluoroscopic outfit is simpler than that necessary for the making of plates. In the acute stage the fluoroscope detects changes from the normal physiologic motion of the lung. In the subacute and chronic cases of chronic adhesive pleurisy, after operation for empyema the Roentgen ray determines the amount of thickening of the pleura and the amount of available lung tissue beneath. These facts are more difficult to determine by ordinary physical examinations.

29. Bayne-Jones, S.: Bull. Johns Hopkins Hosp., 1916, xxvii, 12.
30. Brown, P.: Boston Med. and Surg. Jour., 1915. clxxiii, 804.

It is possible by the Roentgen ray to watch the expansion of the lung damaged by an empyema, and it is especially gratifying to observe the results of treatment.

Gorter[31] reports two cases of interlobar empyema cured by a simple puncture. The first was a child of 15 months who had had fever for five months, following measles and bronchopneumonia. The child had cough constantly and had difficulty in breathing, anorexia and loss of weight. On examination the child was thin, pale and dyspneic. The lungs showed dulness in the right interscapular region which extended downward obliquely to the right and reached the liver dulness in mid-axillary line. Over this area there was tubular breathing and moist râles. The Roentgen ray showed a corresponding shadow. Leukocytes, 24,000. Puncture in the sixth space withdrew, at a depth of 4 cm., 10 c.c. of greenish pus containing pneumococci. Following this there was a rapid recovery and the disappearance of the sign.

The second case was in an infant of 5 months who in the course of pertussis had bronchopneumonia lasting fifteen days. One month later a fever of intermittent type was noted, caused by purulent pleurisy on the right side. Eighty cubic centimeters of pus were withdrawn and a staphylococcus was found. The temperature continued after this, running from 99 to 102 F. Twenty days after the first puncture examination showed a normal lung at the apex and base, but dulness and tubular breathing in the interscapular region. The Roentgen ray showed a shadow which corresponded to this. Von Pirquet negative. Puncture of the sixth space in the scapular line permitted withdrawal of 10 c.c. of pus at a depth of 6 cm. After this the temperature fell gradually and the signs disappeared. Gorter explains the good effects in interlobar effusions by the relaxation of the tension in the sac caused by withdrawing the fluid. These effusions are usually well walled off from the general pleural cavity, and if recognized, the prognosis is good.

Gerdine[32] made a study of pleural effusions occurring during the acute stage of pneumonia. In six out of fifteen cases the fluid was discovered before the crisis. Most of these fluids were sterile, slightly turbid, containing a large number of leukocytes, mostly polymorphonuclear.

He believes that this type of parapneumonic empyema is usually, but not always, of a benign type, that it occurs frequently, and can be demonstrated by the Roentgen ray, physical signs or puncture. It does not alter the course of the pneumonia. True pus is present more rarely and may contain virulent organisms. The degree of virulence of the organisms, determined by animal inoculations, seems to be of prog-

31. Gorter, E.: Arch. de méd. d. enfants, 1916, xix, 317.
32. Gerdine, L.: Am. Jour. Dis. Child., 1915, xi, 33.

nostic value. Surgical interference should only be considered when the organisms found are of high virulence.

It is evident that operation on a child with pneumonia who has a small collection of sterile fluid in the pleural cavity is not justifiable. Operation should be postponed until after the crisis of the pneumonia, unless the amount of pus is large or the organisms are virulent.

Bertoldi[33] reports two cases of pyopneumothorax in infancy. The first was in a child of 3½ years who was found to have signs of pyopneumothorax, was operated on and died. Necropsy showed a foreign body at the bifurcation of the left bronchus, bronchopneumonia and a small abscess, with a perforation of the pleura. The second case was a 15 months' infant who had a pneumococcus infection resulting in pyopneumothorax and death. The diagnosis was made by the Roentgen ray. Bertoldi advises operation as the only hope in these cases, but only in the nontuberculous variety.

In discussing the pneumodynamics of empyema, Cotton[34] says that the intrapleural pressure is determined by the positive pressure in expiration and the negative pressure in inspiration, the elasticity of the lung, lymphatic absorption, and the surface tension of the great pleural surfaces tending to preserve contact of the lung with the chest wall. The last factor is sufficient in itself to prevent collapse of the lung if the pleura is only slightly torn. With larger wounds the lung contracts unless held by adhesions. When a chest full of fluid is opened the lung does not collapse at once. On the contrary, the fluid is expelled in a jet. This is caused by the cough excited by the pleural reflex. The lung usually expands at first but collapses within a few hours. With a proper dressing the lung does not collapse at all, and therefore in a double empyema a proper valve dressing makes it possible to operate on both sides at the same time. The factors determining lung collapse are the previous collapse and lung compression by the fluid, free access of air, respiration with partial closure of the glottis and an accidental valve action at the wound. Cotton believes that any positive pressure apparatus is dangerous, since this is not the proper way to expand the contracted lung.

Proper drainage is the most important factor and is the true way to prevent collapse. It is important to remove the fibrin masses at the time of an operation to prevent plugging of the tube. The rib resection must be one-half to three-fourths of an inch long so that the finger may be inserted and the fibrin swept or fished out. The patient may be turned so that the wound is directed downward, and coughing will

33. Bertoldi, M.: Thèse de Buenos Aires, 1915; ref. in Arch. de méd. d. enfants, 1916, xix, 167.

34. Cotton, F. J.: Boston Med. and Surg. Jour., 1916, cixxiii, 804.

often expel the fibrin. Cotton believes that irrigations are dangerous and should not be used. Drainage is promoted by whatever best aids expansion of the lung. Most cases of empyema get well by accident, because the wet, pus-soaked dressing acts as a fair valve. Cotton uses a square of rubber tissue held taut at the corners by adhesive strips. A little glycerin is run between this and the chest wall. The drainage tube is put through a tight opening in the center of this and negative pressure is applied by means of a column of water which causes a suction through an arrangement of bottles. The water column should not be over 10 inches high, since a stronger suction than this is dangerous and may cause pleural hemorrhage. Blow bottles are very useful to promote expansion of the lung. Decortication operations are rarely necessary.

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